Archives of Neurology and Psychiatry

VOLUME 36 NOVEMBER 1936 NUMBER 5

Appreciations

TO DR. HUGH TALBOT PATRICK FROM HIS PUPILS

We, your former assistants and the associates of your former assistants, are workers in the field of neurology and psychiatry largely through the stimulus you gave us in our formative years. We eagerly sought the opportunity to be inducted into this field by you. We knew that there was little scientific neurology in Chicago before you blazed the way. What the late Christian Fenger did to give young physicians in the Cook County Hospital and the medical schools of Chicago a vision of scientific pathology and surgery you did in your field. Contact with you by students in classrooms and dispensaries and by physicians in hospitals and sick-rooms and in your office was a steady source of fruitful stimulation to them. We who came in closer contact with you not only were inspired by your teaching and your shining example as a clinician but learned to love you for your kindliness and to admire you for your sterling character and your straightforwardness in dealing with people and problems.

We have long desired to give you some token of our affection. Being a small group and realizing that though you have been so much to us you have been much to the medical profession at large, we went to the editors and trustees of the American Medical Association with our plan for a special number of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY to be dedicated to you. As we expected, they responded enthusiastically, all the more because this publication is your child and owes to you much of its high standing in all countries. This number is presented to you in a spirit of love and gratitude.

PETER BASSOE, M.D., Chicago.

HOMMAGE AU PROFESSEUR HUGH T. PATRICK

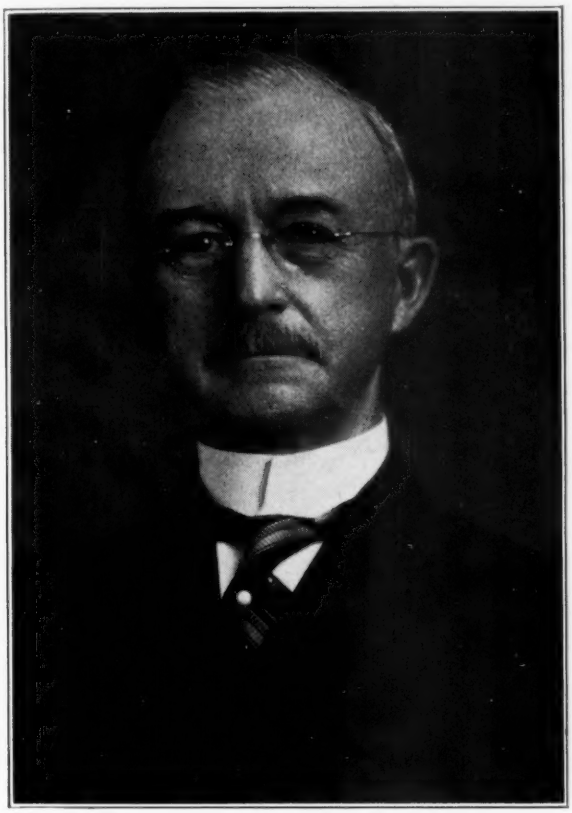
C'est un grand honneur pour moi d'être l'interprète de mes Collègues de la Société de Neurologie de Paris pour adresser au Professeur Hugh T. Patrick, à l'occasion de son jubilé, l'expression de notre sympathie et de notre admiration pour sa belle oeuvre scientifique. Membre correspondant étranger de notre Société, le Professeur Hugh T. Patrick, qui fréquenta autrefois à Paris le service du Professeur Pierre Marie, compte parmi nous de nombreux amis; tous, nous avons suivi sa belle carrière neurologique et avons profité de ses remarquables travaux. Qu'il me soit permis d'exprimer aussi personnellement au Professeur Hugh T. Patrick mes sentiments de très vive gratitude pour l'accueil si charmant que j'ai reçu jadis de lui à Chicago; il m'a fait connaître les éminents neurologistes et les belles Universités de l'Illinois; je conserve de mon séjour á Chicago le plus intéressant et agréable souvenir.

La Société de Neurologie de Paris est heureuse de se joindre aux Sociétés savantes et aux Universités des États-Unis pour exprimer au Professeur Hugh T. Patrick ses très vives félicitations pour son jubilé et ses voeux les plus sincères pour la longue continuation de ses recherches si importantes pour la Neurologie mondiale.

PROFESSEUR GEORGES GUILLAIN, Paris, France.

GREETINGS FROM A CONTEMPORARY

Your sense of humor, friend Patrick, will enable you to accept and appreciate in the proper spirit this series of what I have spoken of elsewhere as "premature obituary notices." And yet there is no harm in hearing from your contemporaries what they think of you, how you



HUGH T. PATRICK

have played your part, what they think of your achievements, your character and your service to mankind. There are few of your professional friends who have known you longer than I. For nearly forty years we have met at the sessions of the American Neurological Association; you were one of the group that included Dercum, Mills, Spiller, Burr, Joseph Collins, Dana, Peterson, Adolf Meyer, Thomas of Baltimore, Harvey Cushing, Tilney, Bassoe and others, each one

of whom has contributed substantially to the structure of American neurology. Let the youngsters of today remember that we of the older group prepared the ground on which they disport themselves, that we built carefully, keeping a solid foundation of facts and rearing clinical neurology on the basis of neuro-anatomy and neuropathology. You were of those who kept their feet on the ground and who, in the Middle West, stood for painstaking scientific investigation, submitting all theoretical speculation and fanciful doctrines to the test of cold logic. You did warm up occasionally and made good use of that mild, shrewd sarcasm of yours, but your criticism was never unkindly and was always worth considering. In fact, the opportunity of greeting you, of having you wink at us with that gracious smile underneath and of listening to your postprandial effusions was one of the chief reasons why I would never miss a meeting of the association these many years. If I were to enumerate your scientific achievements, I should have to refer to your book published in association with Bassoe, to your many excellent articles on arteriosclerosis of the nervous system, chronic progressive hemiplegia, the motor neuron in practical diagnosis (1898), the proper care and treatment of the patient with epilepsy (1907), syphilis of the nervous system (1904) and the factor of fear in nervous disease (1916) and to many other papers showing your wide grasp of the entire neuropsychiatric field. But greater than all these were your founding and direction of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, which has done so much to place American neurology and American psychiatry on the highest scientific level. The pages of the ARCHIVES point proudly to the fact that the American neurologist need apologize to no one and that he is adding substantially to the progress of American medicine. I greet you as former president of the American Neurological Association; let us meet at future sessions to revive and maintain the friendship of these many years. Personally, I greet you as the able neurologist, the right sort of psychiatrist and the colleague of sterling character, admired by many, beloved by all.

BERNARD SACHS, M.D., New York.

PROGNOSIS OF LETHARGIC ENCEPHALITIS IN CHILDHOOD

WILLIAM HEALY, M.D.
BOSTON

In contributing a paper to this volume in token of deep admiration and affection for Dr. Hugh T. Patrick, my thoughts leap back over thirty years, to the time of my early associations with him. At once new developments in neurologic and psychiatric practice come vividly to mind. Nothing stands out more sharply than the fact that since about 1920 society has been confronted with an immensely important disease unknown before then, namely, lethargic encephalitis.

Some of my main chartings in clinical neurology center about a period of work with Oppenheim, that master neurologist in Berlin, from whom Dr. Patrick had learned much and to whom he sent me. In the clinic in Berlin and from Oppenheim's textbook [1] (with which I was already familiar) a comprehensive view of neurologic practice was obtainable as nowhere else. Oppenheim had contributed a monograph on encephalitis, but his picture of the disease, even in cases in which the condition developed during an epidemic of influenza, gave nothing resembling that which has been recognized since the years immediately following the World War. I speak particularly of the clinic at Berlin because European neurologists later had immense experience with lethargic encephalitis; if the disease had been known earlier they probably would have been the ones to recognize its peculiarities. Indeed, it may be remembered that von Economo in 1917 first gave the disease its common name.

This paper will be concerned little, if at all, with the literature on the subject. Hundreds of articles and several volumes have been written on various phases of this disease, which takes such protean forms and is of great social as well as medical import. The symptomatology, the acute phases and the so-called postencephalitic phenomena have been well covered in many publications. Although large numbers of cases appearing during childhood have been observed in institutions and schools abroad and have been reported in some measure, the best source of information on the possibilities of treating these difficult children

From the Judge Baker Guidance Center.

1. Oppenheim, Hermann: Lehrbuch der Nervenkrankheiten für Aerzte und Studierende, Berlin, S. Karger, 1902.

is to be found in the reports by Bond and his colleagues of their work, now unfortunately discontinued, in Philadelphia.

One point has not yet been clearly worked out, that of the prognosis of this disease when it occurs in childhood. At the moment I am greatly interested in this problem for the practical reason that parents, educators and institutional authorities wish to know what to expect and what to plan for. One finds enormously divergent predictions, not only by general practitioners but by pediatricians, neurologists and psychiatrists —prognoses often made with an assurance that is not bred of experience with long observation of cases. It is not a little curious, in consideration of the many reports of well studied cases in which no abnormal somatic findings were shown by available diagnostic methods, that the absence of pathologic signs, particularly in the reflexes or in the spinal fluid content, should be considered a criterion for prediction. One even finds that the absence of abnormal physical findings is sometimes made the basis of an opinion that the patient never suffered from encephalitis. In spite of much evidence to the contrary as, for example, in diseases of the frontal lobes, the medical profession generally seems considerably to be imbued with the idea that organic disease of the brain, especially any possibly continuing disease process, must give evidence of its existence by somatic signs. Lethargic encephalitis in children is a disease which peculiarly affects personality and behavior tendencies and it is concerning these manifestations that one is asked to make a prognosis.

In a clinic such as the Judge Baker Guidance Center there are unusual opportunities for knowing the patients and making observations over long periods. One sees young persons not primarily because they present neurologic or other medical problems; the grist that comes to the mill consists of children presenting disorders of behavior or personality or those who are educationally maladjusted. For the purposes of this paper I have by no means attempted a follow-up study in every case of lethargic encephalitis observed since the foundation of the clinic in 1917. In the light of the diverse findings to be reported, nothing short of the compilation of a huge number of cases would promise statistical validity, and no one in America can deal statistically with any feature of the disease as may Europeans, who have seen hundreds of cases to the dozens in the United States. I have taken merely some illustrative cases in which the patient had been studied at the clinic and the follow-up period now represents at least seven years after the original attack—the range being from seven to eighteen years since the onset of the disease. The outcome to date in these cases and the correlation of the outcome with facts of certain kinds that might naturally have been considered important in prognosis form the essence of this presentation.

HAS RELATIVE SEVERITY OF ONSET ANY IMPORTANCE FOR PREDICTION OF OUTCOME?

In the following case there was no known attack of encephalitis.

CASE 1.—In 1923 a boy aged 12 years began to suffer from extreme disturbances of sleep and showed great changes in personality. Previously he had been normal, active and bright. A physician, who was a close relative and lived near-by, stated that there had been no previous illness; the parents said that two or three weeks prior to the onset of sleeplessness the boy was kept at home for a few days with a cold, but otherwise nothing peculiar was noted. A few months later, when he had shown increasing symptoms, the disease was diagnosed at the neurologic department of a large hospital, and the boy was treated in the outpatient department. No objective signs were discovered except moderate unilateral weakness of the face.

The boy was first seen at the Judge Baker Clinic in 1924 because of difficulties in school and other troubles. By this time he was much worse and presented altogether the most difficult combination of symptoms ever seen at the clinic. Added to almost complete dislocation of sleeping habits there were extreme hyperactivity, destructiveness, yawning, stretching, spitting and whistling habits, ritualistic rubbing of the body and frequent pseudofaintings. The flow of speech was rapid and incoherent. The boy was unmanageable at home and proved extremely troublesome in an institution for children to which he was sent. He had lost much weight and showed greatly lowered circulation; the temperature by mouth was only 95 F. In spite of the hyperactivity he appeared weak. It was noted that the control of mental functions in some ways was good. He did well in psychologic tests; his intelligence quotient was 107, with an even performance. His mental content proved to be exceedingly interesting, for with all the incoherences and contradictions he brought to light many sexual taboos, even voluntarily explaining his hand-washing habits by referring to guilt of masturbation.

The boy was tried in several institutions, including periods of observation at the Boston Psychopathic Hospital. During a few weeks he had severe edema of the legs, although the renal output was normal. During all this time the reflexes and pupillary signs remained normal, although there were occasional variations; it was noted, for example, that the pupils were unequal on one day and equal on the following day.

The boy had to be committed to a state hospital. A year later he was reported as being very difficult on account of untidy habits, extreme destructiveness and hyperactivity. The diagnosis was "psychosis with organic disease, epidemic encephalitis." During the next two years he stayed at the hospital, with occasional visits home, always being returned, however, because he was unmanageable. With all the difficulties of behavior, the mental faculties remained fairly alert. Finally, the family, who were exceedingly patient with the boy, found it possible to retain him at home, and there was gradual improvement, although the prognosis given at the hospital was, naturally, unfavorable.

In 1936 the physician's relative reports that the patient has had the most remarkable recovery of which he had ever heard. At the time of writing, the young man, now 25 years old, is steady and stable. He has long been employed, and, although not working at the level at which his earlier mental capacity would have promised, he is doing well and is reliable. His employers state that he is notably thorough in his work. He shows good judgment in general and seems to be normal mentally. For two years he has attended night school, in an attempt to make up for what he lost educationally. The physician has seen him frequently and has noted no abnormalities except perhaps in his occasional tendency to throw back his head and stretch as he formerly did frequently.

In the following instance the onset was in marked contrast with that in the case just described.

Case 2.—In 1928 a boy aged 13 years suffered from a severe attack of lethargic encephalitis. The diagnosis was clearly evident, and for many months the boy was so ill that the mother said she often prayed that he might die because the physician said he would never be normal. Earlier, he had been regarded as a troublesome lad, who hated school and had habits of petty pilfering, which were out of line with the standards of his somewhat overly good parents. As a small boy he had had temper tantrums.

A fairly accurate history of this boy during the years following the attack was obtainable. No changes in behavior or personality ensued; indeed, it is apparent that in behavior tendencies the boy had considerably improved. Some troubles developed, partially, I am convinced, because of the overprotective attitude taken by the much concerned parents and because of the fact that neighbors in the small home community believed that no patient who had had sleeping sickness could ever be normal. The boy had periods of discouragement due to worries about himself induced by the facts that neighbors and companions always looked askance at him and that he knew that his physician had stated that recovery from encephalitis was unknown.

At the age of 19 the young man was a steady worker; he left school at 16, while in the tenth grade, in order to go to work, partly because the family finances were depleted. He was brought to the clinic because of a rather ordinary bit of misconduct over which the parents made much. In general, even they acknowledged that he showed good attitudes and made good social contacts. He was friendly and met a rather difficult home situation in a fairly manly fashion. Since the illness he had had a moderate amount of trouble with sleeping and persisting frequency of urination. The pupils were normal in size and reactions. The deep reflexes showed no abnormalities, but the abdominal and cremasteric reflexes were absent. There were a fine tremor of the fingers and a slight coarse tremor of the tongue. Physical examination gave otherwise normal results.

On deciding to further his education, he entered a private school a year ago. Finding the course rather difficult, he again began to worry about the after-effects of encephalitis and made an attempt at suicide. Psychiatric treatment and encouragement led him into greater self-confidence. He finished the year with good marks and speedily obtained another position.

A third case illustrates the immensely varying, and in this instance serious, results after a mild, or even unrecognized, onset.

Case 3.—In 1927 a boy aged 11 years was referred from a state hospital clinic to the Judge Baker Clinic because it was not known what to make of him. For about a year he had shown excessive thieving propensities, which took many forms, and had been difficult to manage in school. Great pains were taken to elicit the history of illness and behavior, for the parents said that the present tendencies were out of line with what he had shown earlier and were entirely at variance with the behavior of the siblings. Evidently, there had been a definite change in the boy, beginning at an indefinite period from one to two years earlier. There had been no evidence of illness. All that could be ascertained was that about four years earlier, after an attack of tonsillitis the patient had appeared underactive and "dopey" for about two weeks. Other children in the family were ill with the same trouble at that time, but none exhibited underactivity after the attack. At no time

were there any disturbances of sleep nor did the boy show these signs later. The family expressed the belief that in some way the boy must be sick mentally.

When the boy was seen first, in 1927, there was marked asymmetry in the use of the facial muscles; otherwise he had an active and alert expression. All pupillary signs and reflexes were normal. There was poor mental and motor control in performing psychologic tests, but the intelligence quotient was 95. The patient was hyperactive, overtalkative and distractible. These characteristics, as well as his thieving, made him difficult to manage in school, as well as at home.

Ten months later, in 1928, the family reported that for months he had slightly dragged the right leg and held the right arm in a rigid position. Besides this, he had a habit of continually opening and shutting his right hand and had become clumsy in play. He was even more active and restless. Polyuria had developed. When seen again at this time he was talkative and facetious. The left knee jerk was then more active than the right, but there was no clonus or Babinski sign. The abdominal and cremasteric reflexes were normal, as were also the pupillary reactions. The use of the facial muscles was even more markedly asymmetrical. A few months later he was received into the hospital which had originally referred him to the clinic. A little later they reported scanning speech and grimacing, with gradual mental and physical deterioration. The boy rapidly became worse and died in 1931—six, or possibly eight, years after the onset of the disease.

HAS THE OUTCOME ANY RELATIONSHIP TO THE AGE OF ONSET?

CASE 4.—A boy when about 2½ years of age seemed sleepy and "dopey" for several days. The family physician called twice but stated that there was nothing the matter with the boy so far as he could discover and that the illness did not amount to anything. From that time the family found it almost impossible to control the youngster.

The patient was seen first at the clinic in 1927, when he was 6 years of age. The mother appealed for help to place him somewhere because of his uncontrolled behavior. He did such things as climb on the pantry shelves, set fire to the curtains or light matches anywhere about the house. He stole indiscriminately and seemed to have a mania for undressing little girls. While there was no dislocation of habits of sleep, he was extremely restless at night and overactive in the daytime. Whippings and other forms of discipline accomplished nothing. He was disturbing in the schoolroom and would not go to school unless he was dragged. It was impossible to secure placement for the boy, and he remained at home.

Examination at the clinic revealed that the pupils reacted normally and that all reflexes were normal. The intelligence quotient was 101; the boy worked at the psychologic tests with persistence unless he became fatigued.

The family was obliged to care for the patient, although during the next few years he continued to be troublesome. However, there was gradual improvement, and in 1930 he was reported to be an average boy in behavior and performance at school. Outside the school and home he was occasionally in mischief but was not in any serious affairs, although once or twice he was taken into the juvenile court.

Now in 1936, when 15 years of age, the boy states that he often feels nervous and restless but in general is well. It usually takes him two hours to get to sleep. He is rather irritable and quarrelsome at home, probably partly because he senses inferiority to his siblings, who, though of a poor family, are all ambitious and successful. This is said particularly because his behavior outside the home is normal. He is small for his age but fond of sports. He passed through the eighth grade and at the time of writing is taking a trade course at a high school level.

Physical examination shows hyperactive arm and knee jerks, but no clonus or Babinski sign. The abdominal and cremasteric reflexes are lively. There are well marked fine tremors of the hands, slight tremor of the tongue and of the whole body and marked tremor of the eyelids. Both pupils are slightly ectopic but react properly to light and distance. The patient bites his nails excessively. He is evidently rather euphoric but acknowledges that in his wakeful hours at night he has some anxiety about himself, especially about his school work.

In contrast to the preceding case, in which such steady improvement was shown from the symptomatic educational and conduct standpoints, I may cite the following instance:

CASE 5.—A girl aged almost 14 years, who was first seen in 1936, had suffered from a well defined attack of encephalitis at 6 months of age and from infancy had been regarded as a personality problem. She had spent periods in many hospitals and institutions, including two years with Dr. E. D. Bond and his colleagues in Philadelphia. She was not regarded as acceptable in school or in any institution except a state hospital. At the time of admission to the clinic she was a sweet, affectionate, overfriendly, bright-looking child, thoroughly uninhibited and irresponsible. There had been newspaper publicity because of runaway trips; above everything else she loved to travel.

Examination revealed awkwardness, normal development and some weakness of the facial muscles. The deep and superficial reflexes were hyperactive. No tremors were shown. The right pupil reacted more actively to light than the left, but neither pupil reacted to distance. Intelligence tests gave quotients varying from 81 to 85, but often it was impossible to test the patient because of distractibility. She wandered inquisitively about the clinic and was well controlled only when she was shown some performance tests in which she was interested; then she did really good work. Her school acquirement, naturally, was slight. The parents had spent what to them was a small fortune in trying to do everything possible for her. Perhaps it was the child's affectionate response that made them so desirous to keep her out of a state hospital, where she had already been for one period.

CAN THE OUTCOME, PARTICULARLY THE BEHAVIOR AND SOCIAL ADJUSTMENT, BE CORRELATED WITH THE NEUROLOGIC SIGNS?

CASE 6.—A boy aged 9 years with considerable disturbance in the rhythm of sleep was seen in 1927. He had run away a great deal, had engaged in much thieving and was excessively troublesome everywhere on account of uninhibited behavior. From infancy he had been markedly hyperactive and was always restless at night and sleepy during the day. He had been severely ill with influenza at 3 months of age, at a time when the mother also had influenza. (Since this boy evidently had lethargic encephalitis in 1918, he must have been one of the first in the United States to have the disease.)

Physical examination gave practically normal results, though notes at the clinic stated that the abdominal reflexes, though present, were difficult to elicit. An intelligent aunt, who had observed the boy carefully, said that the pupils were at times like pinpoints; neither at the clinic nor elsewhere had they ever been found other than normally responsive. The intelligence quotient was 96. At the clinic he was extremely restless and uninhibited. He had a habit of yawning, but it was not marked.

He continued to be troublesome at home, in school and to the police. In a correctional school in 1930 physical examination again gave normal results, but the boy

was regarded as incorrigible on account of excitability, running away and fighting; it was thought also that he was somewhat paranoid in feeling that he was unfairly treated. The behavior was regarded as typical of a postencephalitic state. In 1935, after he had again been in court for serious forms of delinquency, neurologic examination gave normal results. The boy, however, was regarded as suffering from psychosis and was committed to a state hospital.

In the preceding case, in which there were no neurologic findings, the patient continued to present difficult problems of personality and behavior. In the next case neurologic signs have developed, but there has been steady improvement from the standpoint of behavior and personality.

CASE 7.—A boy aged 9 years, who was first seen in 1924, had had an acute attack of lethargic encephalitis one year before. He had a high temperature and had slept for about three weeks; the eyes were crossed. There had been an entire change of personality; formerly the boy had been quiet and well behaved, but at the time of examination he often ran away from home, had a bad temper and stole. He had much trouble in getting to sleep and during the daytime was overcome with sleep. He was extremely restless and troublesome.

The physical findings at this time were normal. He was an attractive boy, who on occasion could show good mental and motor control. The intelligence quotient was 135. During the next few years this youngster was seen frequently. He was usually likable, talkative and enthusiastic, though occasionally sulky and petulant. He gradually came to have thoroughly good insight into his difficulties. He made great efforts to exercise self-control, but for a long time he was in many difficulties because of excessive truancy and petty delinquency. He was placed in a foster home for a long period and then was sent to a correctional school, from which he ran away often. For a short period he was in a state hospital in New York where patients of this type were accepted for treatment.

As the years have gone on the boy has shown steady improvement in personality. He is a great reader and has educated himself in astonishing fashion. He has much energy and has secured jobs for himself even during difficult times. He is vivacious and good looking. His ideas and words sometimes almost fall over each other when he is in conversation with friends.

In a recent examination it was found that at rest he held his arms in a somewhat flexed position. With all his vivacity there was a possible suggestion of masklike facies, but it was difficult to be sure that this was not a subjective impression. The general posture was normal, as were also the deep reflexes. There were no tremors and the abdominal reflexes were lively. The left pupil was ovoid, and both pupils were almost fixed, showing only a slight reaction, even to light. These conditions have developed only within recent years. The patient is now 21; in spite of hard family circumstances he is supporting himself well.

WHAT IS THE PROGNOSIS FOR THE DEVELOPMENT OF INTELLECTUAL FUNCTIONS?

It has been said that the result of lethargic encephalitis in infancy is mental defect. Cases 4 and 5, however, give clear evidence of no such result.

In cases of onset at a later age one can find the most widely varying outcomes—steady progress in intellectual development, as in cases 2

and 7, psychosis without dementia, as in case 1, or dementia with rather rapidly developing motor involvement, as in case 3.

CASE 8.—A boy at the age of 6 years had a change of personality after long hospitalization with an illness which was diagnosed scarlet fever and measles. Then followed several years of wild behavior of a typical postencephalitic type, but without disturbances of sleep. Various diagnoses and prognoses were made in different clinics. When about 15 years of age the patient began to show signs of dementia; since then, for the last two years, he has been in a state hospital. At no time, even on recent examination, have any neurologic signs been detected.

The outcome in respect to intellectual function has no necessary relationship to the severity of the illness at the time of onset, as is proved by the contrast between cases 2 and 3.

Other cases could be cited bearing on this feature of the uncertainty of outcome. Earlier statements in the literature that deterioration of intelligence does not take place when the onset of the disease occurs later than infancy are contradicted by the histories in some of the cases at this clinic.

WHAT IS THE PROGNOSIS FOR THE HYPERACTIVITY AND RESTLESSNESS WHICH ARE CHARACTERISTIC OF THE DISEASE IN CHILDHOOD?

Hyperkinetic manifestations tend gradually to disappear; usually they diminish greatly during the second decade of life. It need hardly be mentioned that both early manifestations of difficult behavior and restlessness disappear with the advent of a parkinsonian syndrome. Such an outcome is so well known that no illustrations need be given here. It should be mentioned, however, that this syndrome, with its varying degrees, may or may not be accompanied by dementia. Both outcomes are illustrated in the cases in this clinic. It may be noted that among the cases reported in this series there was only one exception (case 5) to this tendency for hyperactivity gradually to diminish; however, this girl is only 14 years old at the time of writing and one is inclined to believe that she is somewhat less restless than she used to be.

WHAT IS THE PROGNOSIS FOR CONTINUANCE OR BETTERMENT OF THE DISORDERS OF BEHAVIOR?

From the standpoint of social management this is important. The papers of Dr. Bond and his co-workers give much more information on this point than I can offer; the results of the Philadelphia experiment in treatment should be consulted. Nevertheless, from the data in the cases in this series some pertinent conclusions may be drawn.

In case 6, the patient at 18 is still criminalistic; however, he has always lived in neighborhoods in which there is a high rate of delinquency, and he has notoriously been one of the leaders in gang activities.

His experiences in the correctional school have by no means abated his proclivities. It is fortunate for the public welfare that he now has been considered psychotic and has been committed to an institution as such.

In case 5, the attractive little runaway girl has periods of becoming bored with her dull, restricted home life and takes to traveling on trains or on boats. When in the summer, however, she has a chance for wide activities on a relative's farm, she gets into little trouble and is content to stay there.

In contrast to the outcome in most other cases it is remarkable that in case 2 the boy was better behaved after the severe attack of encephalitis at 13 years. In spite of the illness he recovered and was again normally strong and active. Undoubtedly, the outcome in this case was due partly to the fact that he lived in an environment in which there was a high moral tone.

The improvement of behavior in cases 4 and 7, in the course of years, was unquestionably due to lessening of internal stresses. The delinquency ceased as the patient became less restless, active and uninhibited.

The prognosis for behavior depends, then, partly on environmental circumstances, partly on the progress or continuance of the disease itself and partly, perhaps, on growth into greater self-control with increasing maturity.

WHAT IS THE PROGNOSIS FOR GENERAL PHYSICAL HEALTH?

Except in rare instances, as in case 3, one can state that the outlook for general health is good. The possibility of recovery is illustrated in case 1, in which the child passed through a period of severely depleted physical condition and regained full health. In practically all of a dozen patients whom I have recently followed, those in whom the onset of the disease was many years before, the general physical status remains nearly at par, even in those with dementia. Indeed, most of these persons are in good general health.

ARE THERE CLEAR EVIDENCES OF A CONTINUING PATHOLOGIC PROCESS BEING SUBTLY ACTIVE IN SOME CASES?

Surely, the conclusion cannot be avoided that damage to the central nervous system frequently continues long after the acute manifestations of the disease. Even if one regards certain manifestations, such as the parkinsonian syndrome, as being due possibly to secondary degenerative processes, it is difficult to understand how certain other symptoms can be correlated with anything other than a continuing active pathologic condition. The developing hemiparesis and other motor phenomena in case 3 are in point—perhaps also late pupillary changes, such as those in case 7, even if otherwise there has been good recovery. Can these be merely aftermaths of the original damage?

The picture presented in case 1 seems to be that of an active process continuing from an unknown onset for three years or so, with gradual subsidence into complete recovery.

It seems strange now that Bond, in a monograph published in 1926,[2] stated that among the children he studied he found no evidence of a chronic encephalitic process. The argument seems rather to be in favor of the opinion expressed by the Dutch observers, with their immense experience, namely, that a continuing low grade inflammatory process is frequent. Many others have expressed the same belief. One must, however, await better diagnostic methods for the detection of such a subtle pathologic condition.

COMMENT AND CONCLUSIONS

The cases described in this paper belong to a highly selected group. The Judge Baker Guidance Center is brought face to face particularly with the social and educational problems that lethargic encephalitis presents. When one realizes the frequent mildness of onset and the difficulties of early diagnosis, which is often missed, one wonders whether in many cases there has been failure to recognize the disease. There may have been many speedy recoveries.

On the other hand, since one finds indubitable instances of the disease resulting in grave changes in personality and disorders of behavior, although neurologic signs and disturbances of sleep are absent (as in cases 3 and 5), it may well be that the cause of not a few difficult careers is lethargic encephalitis. Does this disease offer a possible etiologic explanation for at least some pathologic personalities and in part for the hyperactivity that is shown in such a large proportion of delinquent children? Certainly, this remains an open question.

The conclusion to be drawn from this review of the later history in cases of lethargic encephalitis is that the offering of a prognosis is unwarranted and may be dangerous, as was evident in case 2. The outcome cannot be foreseen. A wise neurologist will avoid predictions when encountering this disease.

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2. Bond, E. D., and Partridge, G. E.: Post-Encephalitic Behavior Disorders in Boys and Their Management in a Hospital, Am. J. Psychiat. **6**:25, 1926.

CEREBROSPINAL FLUID PRESSURE

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AND
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CHICAGO

VOLUME AND PRESSURE

One of the many observations dealing with physical laws governing cerebrospinal fluid pressure which need clarification is the meaning of the ratio between the removal of cerebrospinal fluid and the subsequent reduction in the cerebrospinal fluid pressure. It is needless for our purpose to quote extensively from the literature. It is sufficient to state that Ayala [1] computed what he called a rachidial quotient by dividing the product of the amount of spinal fluid removed and the resulting pressure by the initial pressure. Later,[2] he modified the quotient, dividing the difference between the initial and the final pressure by the quantity of cerebrospinal fluid removed. This is considered to be a numerical indication of the relative volume of the ventriculosubarachnoid spaces at the time of drainage, the volume of the blood and the elasticity of the meninges. Since Ayala considered the last factor to be relatively constant, he assumed that his quotient was an index of the relative volume of the ventriculosubarachnoid system.

Although possibly empirically this index may have some value clinically (Ayer [3] and Balduzzi [4]), it seemed necessary to us to subject this computation to a careful analysis.

The change in pressure which is produced by the removal of fluid from a container differs according to whether one deals with pressure produced directly by a hydrostatic column or with that impressed on the fluid in a container closed by elastic membranes either by the introduction of additional fluid or by pressure applied through an interposed membrane. In the first instance, as may be readily seen from figure 1, the change in pressure produced by removal of similar amounts

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Read in abstract at the Sixty-Second Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 2, 1936.

1. Ayala, G.: Ueber den diagnostichen Wert des Liquordruckes und einen Apparat zu seiner Messung, Ztschr. f. d. ges. Neurol. u. Psychiat. **84**:42, 1923.

2. Ayala, G.: Die Physiopathologie der Mechanik des Liquor cerebrospinalis und der rachidial Quotient, Monatschr. f. Psychiat. u. Neurol. **58**:65, 1925.

3. Ayer, J. B.: The Human Cerebrospinal Fluid, A. Research Nerv. & Ment. Dis., Proc. **4**:159, 1926.

4. Balduzzi, O.: Evaluation de la pression du liquide céphalo-rachidien dans les méningites séreuses et dans les tumeurs de l'encéphale, Encéphale **19**:83, 1924.

of fluid from containers of equal volume depends on the area of the top of the container; when the area at the top is large the fall in pressure will be less than when it is small, similar amounts being removed. When one deals with a cylinder analogous to the spinal dural sac, when the latter is uppermost in position, that is, with the head down, it may likewise be seen that the change in pressure depends not on the volume of the cylinder but on the cross-sectional area. In a long tube of small diameter the drop in pressure consequent to the removal of a certain amount of fluid will be greater than that in a short cylinder of large diameter, both containing the same amount of the liquid.

In a purely hydrostatic system, therefore, the relation of the change in pressure to the amount of fluid removed is determined by the cross-sectional area of the top of the container.

The study of pressure impressed on a liquid in a container enclosed by elastic walls is comparable to that of the cerebrospinal fluid pressure in animals and man in the horizontal position, when the pressure is the result chiefly of the impression of the

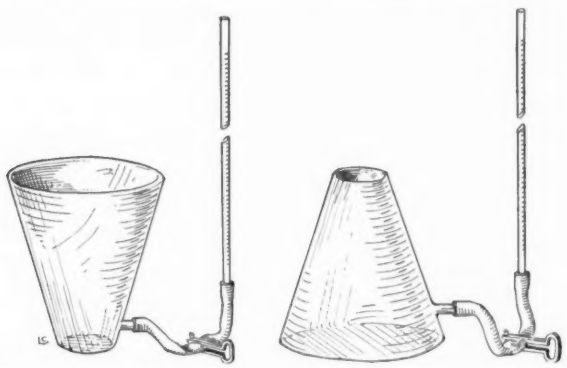

Fig. 1.—Diagram illustrating the relation of the cross-sectional area of the top of a container to the pressure-volume changes.

vascular bed, the production of transudate and secretion into the cerebrospinal fluid and the possible swelling of the neural tissue. This impression acts on the elastic cerebrospinal dural sac, the vessels and the neural tissue, which in turn react to it and are responsible for the ensuing pressure to which the contained fluid is subjected.

To simplify the study of the relation of volume to pressure in a system in which the pressure is produced by the production of fluid, it is necessary only to construct two cylinders closed at one end and covered at the other with elastic membranes of exactly the same size and character. The cylinders are constructed so that when a head of pressure of 95 cm. of water is applied and then locked in the cylinders, each cylinder will contain 130 cc. of water. One cylinder has a diameter of 42 mm. and the other of 25 mm. (fig. 2).

In the cylinder with a rubber membrane of 42 mm. diameter, the absolute pressure [5] dropped from 95 to 89.8 cm. when 1 cc. of fluid was removed, whereas

5. By relative pressure we mean the height, expressed in centimeters, to which fluid rises in an open manometer, and by absolute pressure, the height, expressed in centimeters, above the cistern needle of the level of fluid in the manometer when the flow into the manometer is prevented by elevating the manometer, so that the level of the fluid remains constantly at the original level.

in the cylinder with a diameter of 25 mm. the absolute pressure dropped from 95 to 37.8 cm. when 1 cc. was removed. It is obvious that the ratio of the drop in pressure to the amount of fluid removed is related to the area and the elasticity of the enclosing elastic membranes and not to the amount of fluid in the container.

The quotient obtained by dividing the difference between the initial and the final pressure by the quantity of fluid removed gives 5.2 in the case of the 42 mm. cylinder and 57.2 in that of the 25 mm. cylinder, the original volume being 130 cc. in each instance.

When in such a model, filled at atmosphere, a certain head of pressure is impressed on its contents, a deformation in the limiting elastic membrane occurs, permitting the inflow of an additional amount of fluid, which is constantly related for each model to the amount of pressure. When a head of 10 cm. of pressure was imposed on the contents of a cylinder with a diameter of 25 mm. a deformation of the membrane occurred, allowing 0.2 cc. to flow in, whereas in a cylinder with a diameter of 42 mm. 2.19 cc. flowed in and in a cylinder with a diameter of 106 mm. 51.1 cc. was accommodated. When these amounts were removed, respectively, from the several cylinders, the pressure in each dropped to zero, and when the same amounts were reintroduced the pressure in each rose to 10 cm.

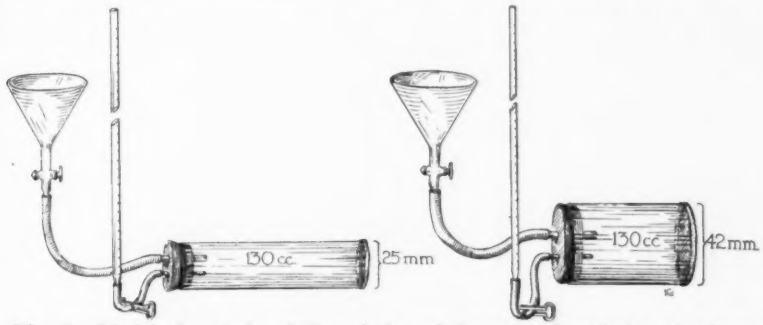

Fig. 2.—Models for study of the relation of the area of enclosing elastic membranes to pressure-volume changes.

When the experiment is reversed and cylinders of the same diameter but of different volumes are used, it is found that the same amount of pressure produces deformations of similar amounts in the two cylinder membranes and that removal of the same amount of fluid from each cylinder when under the same head of pressure produces equal drops in pressure.

It is thus seen that the ratio of the amount of fluid removed to the change in pressure is related only to the fluid which occupied the space produced by the deformation of the limiting elastic membrane and not to the total amount of fluid enclosed in the container. The amount of deformation is related to the area and elasticity of the membrane.

This holds true for the condition when the pressure is impressed on the contents of such a cylinder through a membrane interposed between the head of pressure and the contents of the cylinder, as in the case of a vascular bed acting on cerebrospinal fluid enclosed within a possibly elastic cerebrospinal dural sac. Such a condition would be approximated by a model in which a head of pressure is put on a rubber membrane covering a tambour, which is placed in a cylinder closed at one end by a rubber membrane and filled with water (fig. 3).

Here the amount of pressure developed in the cylinder would depend on the deformation of the tambour, which is related to its area and elasticity, as well as the elasticity and area of the cylinder membrane.

When the tambour acted against atmospheric pressure that is, lay free in the air, a head of pressure of 40 cm. of water produced a deformation permitting 3.2 cc. to flow into the tambour. When the tambour membrane acted against the membrane of the cylinder, which was filled with water, a head of 40 cm. of water produced a deformation allowing only 2.5 cc. of fluid to flow into the tambour. When in the latter instance after 2.5 cc. of fluid had flowed into the tambour the head was closed off, the same amount removed from the cylinder and the head again opened, an additional 0.49 cc. of water flowed into the tambour. Although when in such a system a static state exists the ratio of the removal of fluid to the resulting pressure is related to the area and elasticity of the cylinder membrane, the ratio is related to both the cylinder and the tambour membrane when, as in the experiment just described, a dynamic state exists. In other words, when a contained head of pressure is put on the tambour membrane (analogous to the vascular bed) the area and elasticity of both the limiting membrane of the cylinder (dural sac, vessels, etc.) and that of the tambour (vascular bed, etc.) are related to the ratio of volume to pressure.

It is conceivable that under certain pathologic conditions there may be a modification of this rule. When, for example, the cerebrospinal dural sac has

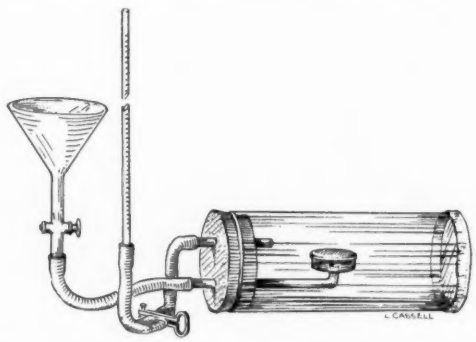

Fig. 3.—Model for study of the relation of volume to pressures impressed through an elastic membrane.

lost its elasticity and acts as a rigid membrane and the distensibility of the vascular bed has almost reached its fullest extent, so that the removal of a small amount of fluid about it does not result in a reciprocal further distention, the pressure may drop considerably and no true ratio exist between the removal of fluid and the drop in pressure.

When, because of distention of the vascular bed and the swelling of the neural tissue, the cerebrospinal fluid is displaced, so that its amount is reduced, and a sufficient amount remains to distend the cerebrospinal dural sac, the ratio of the fluid removed to the reduction of pressure must still be related to the area and elasticity of this sac. If only a part of the sac is in contact with the fluid, the area and elasticity of that part are related to the volume-pressure ratio.

A balloon within a cylinder filled with water and covered at one end by a rubber membrane was distended by a head of pressure of 50 cm. of water, so that part of the balloon was in contact with the cylinder wall. The head then was shut off and the pressure within the cylinder reduced to atmosphere. The head was then raised 10 cm. higher and again shut off; the pressure within the cylinder was found to read 7.5 cm. of water. When 3 cc. of water was removed

from the cylinder the pressure dropped to zero. If then some of the water in the cylinder was displaced by introducing a number of glass beads, care having been taken not to let them impinge on the surface of the balloon at the bottom, so that the quantity of fluid was reduced from 116 to 88 cc., an additional head of 10 cm. of water produced a pressure of 7.4 cm. Removal of 2.9 cc. of water reduced the pressure to zero.

Quotients obtained by dividing the difference in pressures by the difference in volumes, or its reciprocal, differ greatly according to the position of the animal and to whether the pressure is to a greater degree due to impression of a vascular bed, as in jugular compression, or to hydrostatic force, as in tilting, and, finally, to whether fluid is extracted from the cerebrospinal system or is allowed to be dislocated into a manometer. The last-mentioned factor can readily be studied in a model of a 27 cm. tube covered at the top with a 35 mm. membrane (fig. 4). When the end with the membrane is uppermost and the 11 mm. manometer is opened, the relative pressure is 6.9 cm. of water, and the fluid displaced is 0.561 cc.,

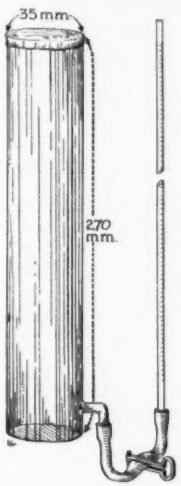

Fig. 4.—Model permitting comparison of volume-pressure changes when fluid is displaced into a manometer and removed from a container.

the absolute pressure being 18.5 cm.; the quotient of $\frac{dP}{dV}$, the difference in pressures divided by the difference in volumes, or $\frac{11.6}{0.561}$, equals 20.68. Then, the original state being restored, 0.561 cc. is removed from the cylinder by a syringe. The relative pressure becomes 3.3 cm., and the absolute pressure, 13.5 cm. The difference in the absolute pressures is 5 cm., and this divided by 0.561 gives a quotient of 8.911, less than one-half the other. If one divides the difference in relative pressures by 0.561, the quotient is 6.417. The results obtained by removing fluid from the cylinder and those obtained by allowing it to run into a manometer are entirely different, because the mechanics are different. This will be studied fully in a later section dealing with elasticity. It is necessary only to refer to the protocol of one animal to illustrate the various quotients obtained under different conditions, and descriptions of many of the experiments on animals may be left to a later section.

Cerebrospinal fluid pressures were measured through a needle in the cistern of an animal under pentobarbital sodium anesthesia. With the animal in the horizontal position the relative pressure was 9 cm. of water and the absolute pressure 12 cm. The quotient obtained by dividing the amount of fluid displaced into the manometer by the difference in pressures was 0.081. On jugular compression the relative pressure rose to 21.2 cm. and the absolute to 31 cm.; the quotient $\frac{dV}{dP}$ was 0.0584.

Then when the animal was tilted 30 cm. with the head down, the relative pressure rose to 25.5 cm. and the absolute to 49 cm. The quotient $\frac{dV}{dP}$ was 0.037, or about one-half that for the horizontal position. When the animal was restored to the horizontal position and 0.5 cc. of fluid was removed from the cistern, the relative pressure dropped to 15.8 cm. and the absolute to 23.5 cm. The quotient $\frac{dV}{dP}$ was 0.0697. Not only were various differences of pressure present in each instance but many factors were at work which cannot be explained or even indicated by this simple quotient. From his observations in man, Masserman[6] concluded that Ayala's formula cannot be applied with accuracy to determine the relative sizes of the ventriculomeningeal system in a series of normal patients and that it may be misleading when applied to patients with complex pathologic alterations in the brain and meninges. Our studies on models and animals afford a clue to the reason for the inaccuracy.

Comment.—The change in pressure resulting from the removal of cerebrospinal fluid is related to the area and elasticity of the enveloping membranes of the cerebrospinal fluid system and, in exceptional cases, to the limitation of distensibility of the vascular bed.

Since the area and elasticity of the enveloping membranes of containers are related to the ratio of the fluid removed to the change of pressure, these factors being unknown, any quotient obtained by the division of the amount of fluid removed by the differences in pressure, or its reciprocal, will be only an arbitrary figure, perhaps of some clinical use but in no way indicative of the amount of fluid present in the cerebrospinal fluid system.

CEREBROSPINAL ELASTICITY

Evidence of the existence of an important relationship between the dislocation of cerebrospinal fluid and its pressure was presented by Weed, Flexner and Clark in 1932.[7] Since, they have extended their studies in the definition of this relationship.[8]

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6. Masserman, Jules H.: Cerebrospinal Hydrodynamics: IV. Clinical Experimental Studies, Arch. Neurol. & Psychiat. **32**:523 (Sept.) 1934.

7. Weed, L. H.; Flexner, L. B., and Clark, J. H.: The Effect of Dislocation of Cerebrospinal Fluid upon Its Pressure, Am. J. Physiol. **100**:246, 1932.

8. Flexner, L. B.; Clark, J. H., and Weed, L. H.: The Elasticity of the Dural Sac and Its Contents, Am. J. Physiol. **101**:292, 1932. Weed, L. H., and Flexner, L. B.: Cerebrospinal Elasticity in the Cat and Macaque, ibid. **101**:668, 1932. Flexner, L. B., and Weed, L. H.: Note on Cerebrospinal Elasticity in a Chimpanzee, ibid. **105**:571, 1933.

They found from their tilting experiments that the difference in the volumes of displaced fluid, i. e., into an open manometer, has a definite relationship to the difference in pressure change. This relationship is expressed by the fraction $\frac{dV}{dP}$, in which dV represents the difference in volume change when the animal is tilted from a horizontal to the head-down or the tail-down position, with no external displacement of fluid (absolute pressure), and the actual change in volume, expressed in cubic centimeters, in any of the open end manometers (relative pressure), and in which dP represents the difference between the relative and the absolute pressure on tilting. This fraction $\frac{dV}{dP}$, which in dogs of uniform size was found to have an average value of 0.17, was also derived by using the difference in the changes in pressure with any two open end manometers of different bores for comparison with the corresponding difference in volumes. It was pointed out that the fraction $\frac{dV}{dP}$ is related to the general physical formula for an elastic system. In this formula for the coefficient of the "volume elasticity," E, the stress is the change in pressure, dP, and the strain is the change in volume, dV, divided by the original volume, V; i. e., E, the coefficient of elasticity, $= \frac{\frac{dP}{dV}}{V} = \frac{dP}{dV} v$. The authors stated that "in any one animal, the normal volume V does not seem to vary appreciably as any theoretical or actual dislocation of cerebrospinal fluid may be entirely or partially compensated by a reciprocal readjustment within the vascular channels," and they expressed the belief that "one would expect the fraction $\frac{dV}{dP}$ to remain constant as long as the animal remained in good condition with normal responses." In most of their experiments they stated that they found this condition to hold true.

Later,[8a] in a series of experiments, they obtained the value of this fraction for an individual animal and then measured the volume of the animal's intradural, cranial and spinal contents, which permitted them to derive the coefficient of elasticity in dynes per square centimeter by substitution in the formula $E = V \frac{dP}{dV} = V / \frac{dV}{dP}$.

For our purpose in this communication, it is not necessary further to analyze their data, some figures of which will be referred to later. It is important to make several observations. In speaking of E, the coefficient of elasticity, they stated: "[It] is a general elastic coefficient for the system as a whole and should not be confused with the coefficient of linear stretch (Young's Modulus) of the membranes and owing to the complexity of the system cannot be related to it."

Since the terms coefficient of elasticity and modulus of elasticity are recognized as related to definite constants and are computed by rigid mathematical formulas, whether in the form of a bulk modulus, a shear

or Young's modulus of stretch, or bending, it appears to us that if this general coefficient of elasticity is a constant it could be defined even for this "system as a whole."

The equation $dP/\frac{dV}{V}$ is the bulk modulus. This, of course, is related to the changes in volume which occur in a body which may be compressed but the shape of which remains unchanged by variations in pressure. In such a case one measures the compressibility of the mass, which, if applied to the cerebrospinal contents, would be the compressibility of the fluid, blood and nerve tissue.

In the course of experiments designed to study other factors of cerebrospinal fluid pressure we had occasion to study the relation of

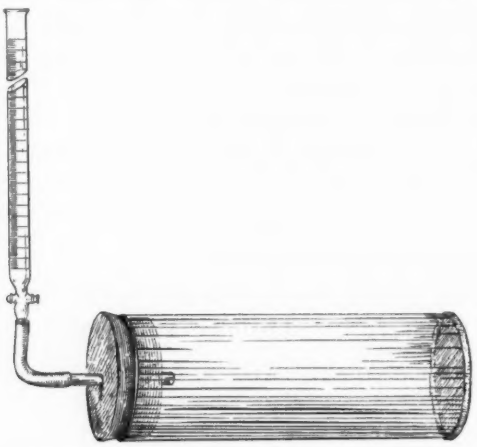

Fig. 5.—Model for study of the flow of fluid into a container as produced by successive increases in the head of pressure.

changes in pressure to removal of fluid in various positions and under various conditions and to dislocation of fluid into open end manometers. When our figures were completed according to the equation that Weed and his associates described, they showed so many differences that we thought it would be of interest to attempt to discover some of the reasons for these variations.

It is of interest, first, to note what occurs in the simplest form of a model (fig. 5). A head of pressure is put on fluid contained in cylinders which are closed at one end by rubber membranes 25, 38, 42 and 106 mm., respectively, in diameter and 0.27 mm. in thickness. When a cylinder with a 38 mm. membrane is used, as shown in table 1, it is seen that with successive rises of pressure there is a diminishing inflow of fluid as the membrane becomes stretched more and more to a certain point, beyond which there is an increased inflow, which at high pressures becomes very great. Beyond this point of elastic limit we believe that an elastic viscous flow occurs, which is responsible for this disproportionate change in volume.

TABLE 1.—*Amounts of Fluid Displaced into Cylinders of Different Diameters but of the Same Capacity with Increasing Heads of Pressure and Determinations of the Resulting* $\frac{dV}{dP}$ *and* $\frac{dP}{dV}$ *V*

	25 Mm. Membrane				38 Mm. Membrane					42 Mm. Membrane				106 Mm. Membrane			
Head of Pressure	Fluid Displaced Into Cylinder	Successive Differences in Fluid Displaced	$\frac{dV}{dP}$	$\frac{dP}{dV}$ V	Fluid Displaced Into Cylinder	Successive Differences in Fluid Displaced	$\frac{dV}{dP}$	$\frac{dP}{dV}$ V	Reciprocal of $\frac{dP}{dV}$ V	Fluid Displaced Into Cylinder	Successive Differences of Fluid Displaced	$\frac{dV}{dP}$	$\frac{dP}{dV}$ V	Fluid Displaced Into Cylinder	Successive Differences of Fluid Displaced	$\frac{dV}{dP}$	$\frac{dP}{dV}$ V
5	....	....	.....	.....	0.78	0.78	0.156	833	.....	....	....	.....	.....	.....	.....	.....	....
10	0.20	0.20	0.0200	6,500	1.56	0.78	0.156	838	0.2000	2.19	2.19	0.219	594	51.10	51.10	5.110	25.4
15	....	....	.....	.....	2.28	0.72	0.152	914	0.1920	....	....	.....	.....	.....	.....	.....	....
20	0.44	0.24	0.0220	5,425	3.00	0.72	0.150	919	0.1890	3.60	1.41	0.180	937	101.36	50.26	5.068	36.0
25	....	....	.....	.....	3.62	0.62	0.145	1,073	0.1820	....	....	.....	.....	.....	.....	.....	....
30	0.69	0.25	0.0230	5,218	4.28	0.62	0.142	1,075	0.1790	4.80	1.20	0.160	1,113	172.36	71.00	5.745	33.0
35	....	....	.....	.....	4.86	0.58	0.139	1,157	0.1740	....	....	.....	.....	.....	.....	.....	....
40	0.92	0.23	0.0230	5,682	5.54	0.68	0.139	992	0.1710	5.95	1.15	0.149	1,172				
45	....	....	.....	.....	6.16	0.62	0.137	1,093	0.1720	....	....	.....	.....				
50	1.09	0.17	0.0218	7,701	6.86	0.70	0.135	974	0.1730	7.20	1.25	0.144	1,079				
55	....	....	.....	.....	7.58	0.72	0.137	951	0.1740	....	....	.....	.....				
60	1.29	0.20	0.0215	6,555	8.28	0.70	0.138	983	0.1740	8.62	1.42	0.144	966				
65	....	....	.....	.....	9.21	0.93	0.140	745	0.1780	....	....	.....	.....				
70	1.47	0.18	0.0210	7,293	10.26	1.05	0.146	663	0.1869	10.53	1.91	0.150	725				
75	....	....	.....	.....	11.56	1.30	0.154	556	0.1950	....	....	.....	.....				
80	....	....	.....	.....	13.38	1.82	0.167	389	0.2130								
85	....	....	.....	.....	17.12	4.74	0.200	151	0.2610								

The small differences we have noted are certainly not the result of accident or error but are related to a definite law that at different degrees of distention of an elastic membrane the same change in pressure produces different deformations. With the same membrane one obtains diminishing and then increasing dV/dP quotients. The deformation, then, is the result not of pressure alone but of pressure per unit area, or thrust. This is seen in the case of a balloon partially constricted at its upper one-third by a ligature. When a head of pressure is put on the contents of such a balloon, the part with the larger area distends until it is rigid and large; the smaller part seems softer and is but little distended (fig. 6). That this is true is also seen by comparing in table 1 the amounts of fluid which run into the cylinders of different diameters as the result of increasing heads of pressure; the larger the diameter the more fluid runs in. Again, in this table one sees that the dV/dP quotient varies for the different cylinders with the same pressure, for instance, at a pressure of 20 cm., 0.022 for the 25 mm. cylinder, 0.15 for

Fig. 6.—Model illustrating the relation of the degree of thrust to the area.

the 38 mm. cylinder, 0.18 for the 42 mm. cylinder and 5.068 for the 106 mm. cylinder, parts of the same sheet of rubber dam being used for the membrane. The same variation is seen in the values for $\frac{dP}{dV}$ V. Weed has called attention to this variation in a model, stating that in this instance values for dV/dP of different magnitude were derived when containers of different diameter were used, "yet these factors were remarkably constant for each experiment." This is true, but unless one can determine the area of the elastic membrane at work at any particular moment when a pressure is taken, we believe that the dV/dP quotient is meaningless, as further analysis seems to show. It is immediately obvious that for a quotient to have meaning in this system other factors must be considered.

The possible relationship of the original volume in a container can easily be studied if one uses two cylinders so constructed that when under 70 cm. of pressure each contains 130 cc. of fluid; the one has a diameter of 25 mm., the other, of 42 mm. (fig. 7).

The amount of fluid running into the 25 mm. cylinder to make the 130 cc. was 1.49 cc., and that into the 42 mm., 10.64 cc.

When we repeated the experiment, using cylinders the volume of which was 130 cc. before a head of pressure was put on, we found that with the same pressure 1.47 cc. had entered the 25 mm. cylinder and 10.53 cc. the 42 mm. With the use of cylinders of dissimilar volume but of the same diameter the same amount of water always ran into the cylinder at the given pressure.

Finally, referring to table 1, one sees that if the original total of volume is used as a factor in determining a possible coefficient of elasticity (E) by the equation $\frac{dP}{dV}V$, the quotients are no more constant than the fraction $\frac{\Delta P}{dP}$. Therefore, in a system bounded partly or wholly by elastic walls changes in the amount of fluid as related to the changes in pressure bear a relation not to the original amount of fluid but to the amount which runs in as the result of deformation of the elastic membranes and are definitely related to the area of the elastic membrane.

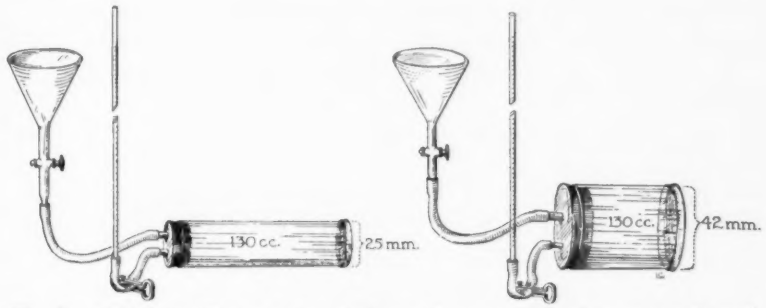

Fig. 7.—Models to illustrate that similar volume-pressure changes are dependent on the area of the limiting elastic membranes and not on the volume of the containers.

Weed and his co-workers said that "were the elasticity of an imperfect character, varying with different loads and with different amounts of dislocation of fluid, no constant fraction could possibly be obtained." [7]

Realizing from the experiments with the deformation of rubber membranes not only that a variation in the displacement of fluid occurs in relation to the degree of distention but that rubber is imperfectly elastic, we investigated the condition in animals. It was natural here to find only the confirmation of an accepted opinion—that the elasticity of biologic substances, such as muscle, differs from that of a perfectly elastic substance. We shall refer to an experiment of each kind: first, the change in pressure that occurs when, after the cerebrospinal fluid has been permitted to escape at atmosphere through a cisternal puncture, similar amounts of fluid are introduced successively and the pressures in an open and an absolute manometer are measured; second, the changes in pressure consequent to the successive removal of constant quantities

of fluid, and, last, the amount of fluid which enters the cerebrospinal cavity when, after the cerebrospinal fluid has been permitted to escape at atmosphere from a cisternal puncture, fluid is introduced by elevating a head of pressure 1 cm. at a time.

In the last instance, we found that in the animal there was no constant relation between the height of pressure and the amount of fluid introduced. At first there was an increase in the deformation with successive pressures and then a diminution, until, finally, with higher pressures little deformation occurred with successive rises in pressure. The curve in this instance differed from that for the rubber membrane, in which successive rises of pressure at first caused diminished and then greater deformations, but the variability of the relationship between volume and pressure was obvious in both instances (table 2).

TABLE 2.—*Displacement of Fluid into the Cistern by Successive Increases in the Head of Pressure and the Resulting* $\frac{dV}{dP}$

Head of Pressure, Cm.	Fluid Displaced into Cistern, Cc.	Successive Changes in Fluid Displaced, Cc.	$\frac{dV}{dP}$
1.1	0.190	0.190	0.173
2.0	0.235	0.045	0.128
3.0	0.400	0.165	0.133
4.25	0.575	0.175	0.135
5.0	0.760	0.185	0.152
6.0	1.090	0.330	0.182
7.0	1.410	0.320	0.201
8.0	1.845	0.435	0.231
9.0	2.085	0.240	0.232
10.0	2.265	0.180	0.2265
11.0	2.475	0.210	0.225
12.0	2.605	0.130	0.217
13.0	2.660	0.055	0.200
14.0	2.800	0.140	0.200
15.0	2.880	0.080	0.192
15.8	2.890	0.010	0.183
16.7	2.925	0.035	0.175
17.7	2.950	0.025	0.167
18.5	2.980	0.030	0.161
20.7	3.020	0.040	0.146

* The animal was under pentobarbital sodium anesthesia, and 3 cc. was removed from the cistern to reduce the pressure to zero.

Naturally, the $\frac{dV}{dP}$ quotient, in which dV is the amount of fluid running into the system and dP the pressure of the head, varied at different pressures in the first and second phases and, particularly, in the last phase with high pressures. When one studies a complete series of readings, one realizes that readings taken at one or two positions, or only a few readings, may show small differences in the $\frac{dV}{dP}$ quotients that may be taken to be variations in elasticity, errors and the like but which really are differences in the quotients with different states of pressure relationships and are not accidental.

In an animal in which the cisternal pressure was 11.8 cm. of water, a quantity of physiologic solution of sodium chloride was introduced to raise the pressure

to 37.5 cm. Then 0.5 cc. quantities were successively removed, and the absolute pressure was recorded. The first 0.5 cc. reduced the pressure 14 cm., dV/dP, 0.0357; the second 3.5 cm., dV/dP, 0.143; the third, 2.5 cm., dV/dP, 0.2; the fourth, 1.2 cm., dV/dP, 0.417, and the fifth, 1.5 cm., dV/dP, 0.333.

While one is concerned here with large changes in pressure and volume, the changing dV/dP is nevertheless observed.

When, having allowed the cerebrospinal fluid to escape at atmosphere in another animal until the pressure fell to zero, we successively introduced 0.5 cc. of physiologic solution of sodium chloride, we found that the first 0.5 cc. produced a rise of 3.1 cm. in pressure, $dV/dP = 0.161$; the second, 2.5 cm., $dV/dP = 0.2$; the third, 1.6 cm., $dV/dP = 0.312$; the fourth, 2.6 cm., $dV/dP = 0.192$; the fifth, 2.2 cm., $dV/dP = 0.227$; the sixth, 4.1 cm., $dV/dP = 0.122$, and the seventh, 8.7 cm., $dV/dP = 0.0575$.

TABLE 3.—*Comparison of the Quotients Obtained by Dividing the Amount of Fluid Displaced into a Container by the Increase of Pressure and by Using Determinations of Relative and Absolute Pressures in Open Manometers*

Pressure, Cm.	Displacement of Fluid into Cylinder, Cc.	Successive Changes in Volume in Cylinder, Cc.	$\frac{V}{P}$	Pressure in Relative Manometer, Cm.	Pressure in Absolute Manometer, Cm.	Volume in Manometer, Cc.	$\frac{dV^*}{dP\dagger}$	$\frac{dP}{dV}$ V
5	0.78	0.78	0.156	4.20	5.0	0.1034	0.141	922
10	1.56	0.78	0.156	8.95	10.0	0.2416	0.230	554
15	2.28	0.72	0.152	12.00	14.7	0.3240	0.120	1,096
20	3.00	0.72	0.150	16.30	20.0	0.4401	0.118	1,121
25	3.62	0.62	0.145	19.90	24.7	0.5373	0.112	1,188
30	4.28	0.62	0.142	23.85	30.0	0.6439	0.106	1,261
35	4.86	0.58	0.139	27.70	35.0	0.7479	0.102	1,316
40	5.54	0.68	0.139	31.90	40.0	0.8613	0.105	1,284
45	6.16	0.62	0.137	35.80	45.0	0.9666	0.105	1,291
50	6.86	0.70	0.135	40.30	50.1	1.0881	0.111	1,226
55	7.58	0.72	0.137	44.60	55.1	1.2042	0.115	1,190
60	8.28	0.70	0.138	48.80	59.9	1.3176	0.118	1,166
65	9.21	0.93	0.140	54.30	65.1	1.4661	0.135	1,025
70	10.26	1.05	0.146	59.70	70.1	1.6119	0.155	900
75	11.56	1.30	0.154	65.30	74.9	1.7631	0.183	758
80	13.38	1.82	0.167	72.90	79.9	1.9683	0.281	504
85	17.12	3.74	0.201	80.60	84.0	2.1762	0.640	224

* Fluid in relative manometer.
† Relative and absolute manometers.

The curve here is the reciprocal of that for the amount of fluid which runs in at successive increases of pressure and at first diminishes and then increases, dV/dP varying throughout, although here, too, the pressures generally exceeded those seen with the animal in the horizontal position, except on compression of the jugular veins. In the first series of experiments about thirteen readings were within the usual range of normal pressures with the animal in the horizontal position. We believe on the basis of this finding that, without other factors being known, the dV/dP quotient is not an index of the elasticity of the cerebrospinal contents.

In addition to this method for computing the quotient, dV may represent the amount of fluid in an open manometer when dP is the difference between the pressure in the open manometer and that in an absolute

TABLE 4.—*Comparison of* $\frac{dV}{dP}$ *and* $\frac{dP}{dV}V$ *Computed by Using Relative and Absolute Pressures in Open Manometers with Containers of Different Diameters*

Head of Pressure	25 Mm. Membrane					38 Mm. Membrane					42 Mm. Membrane					106 Mm. Membrane				
	Relative Pressure, Cm.	Absolute Pressure, Cm.	Manometric Volume, Cc.	$\frac{dV^*}{dP}$	$\frac{dP}{dV}V$	Relative Pressure, Cm.	Absolute Pressure, Cm.	Manometric Volume, Cc.	$\frac{dV^*}{dP}$	$\frac{dP}{dV}V$	Relative Pressure, Cm.	Absolute Pressure, Cm.	Manometric Volume, Cc.	$\frac{dV^*}{dP}$	$\frac{dP}{dV}V$	Relative Pressure, Cm.	Absolute Pressure, Cm.	Manometric Volume, Cc.	$\frac{dV^*}{dP}$	$\frac{dP}{dV}V$
5	....	....	.....	.....	.....	4.20	5.0	0.1034	0.141	922	....	....	.....	.....	.....	....	....	.....	.....	...
10	3.70	7.3	0.0099	0.0280	4,643	8.95	10.0	0.2416	0.230	554	8.0	9.6	0.0216	0.1350	963	9.5	10.3	0.2585	0.324	401
15	....	....	.....	.....	.....	12.00	14.7	0.3240	0.120	1,096	....	....	.....	.....	.....	....	....	.....	.....	...
20	7.75	16.8	0.2093	0.0230	5,661	16.30	20.0	0.4401	0.118	1,121	15.9	19.7	0.4293	0.1130	1,169	20.5	20.6	0.5535	5.535	33
25	....	....	.....	.....	.....	19.90	24.7	0.5373	0.112	1,188	....	....	.....	.....	.....	....	....	.....	.....	...
30	12.10	29.6	0.3276	0.0187	7,000	23.85	30.0	0.6439	0.106	1,261	23.7	29.7	0.6699	0.1120	1,193	30.1	30.4	0.8127	2.709	85
35	.....	....	.....	.....	.....	27.70	35.0	0.7479	0.102	1,316	....	....	.....	.....	.....	....	....	.....	.....	...
40	14.90	38.8	0.4013	0.0170	7,688	31.90	40.0	0.8613	0.105	1,284	31.5	39.8	0.8505	0.1020	1,321	....	....	.....	.....	...
45	.....	....	.....	.....	.....	35.80	45.0	0.9666	0.105	1,291	....	....	.....	.....	.....	....	....	.....	.....	...
50	17.90	46.8	0.4833	0.0167	7,840	40.30	50.1	1.0881	0.111	1,226	39.8	49.6	1.0746	0.1097	1,240	....	....	.....	.....	...
55	.....	....	.....	.....	.....	44.60	55.1	1.2042	0.115	1,190	....	....	.....	.....	.....	....	....	.....	.....	...
60	21.40	58.7	0.5778	0.0155	8,457	48.80	59.9	1.3176	0.118	1,166	48.8	59.5	1.3176	0.1230	1,115	....	....	.....	.....	...
65	.....	....	.....	.....	.....	54.30	65.1	1.4661	0.135	1,025	....	....	.....	.....	.....	....	....	.....	.....	...
70	23.90	67.8	0.6453	0.0147	8,931	59.70	70.1	1.6119	0.155	900	59.1	69.8	1.5957	0.1490	930	....	....	.....	.....	...
75	.....	....	.....	.....	.....	65.30	74.9	1.7631	0.183	758	....	....	.....	.....	.....	....	....	.....	.....	...
80	.....	....	.....	.....	.....	72.90	79.9	1.9683	0.281	504	....	....	.....	.....	.....	....	....	.....	.....	...
85	.....	....	.....	.....	.....	80.60	84.0	2.1762	0.640	224	....	....	.....	.....	.....	....	....	.....	.....	...

* $\frac{dV}{dP}$ expresses the relationship of the difference in volumes and the difference in pressures of the relative and the absolute manometer. The manometric bore was 0.027 sq. cm.

manometer. The quotient may also be measured by using two manometers of different bores, dV being the difference in the amounts of fluid in the two manometers and dP the difference in the heights of the columns. These methods have been used interchangeably, with the animal both in the horizontal and in the vertical position.

Theoretically, the difference between the pressure in an open manometer and the absolute pressure is the amount of pressure necessary to push back into a container or the cerebrospinal system the amount of fluid which flows into the open manometer. If the enclosing membranes were perfectly elastic and the area did not change with different degrees of distention, one could obtain a constant relation between the pressure and the volume. It has been seen that there is a change in the value for dV/dP when dV represents the amount of fluid which enters a container and dP the pressure, as the pressure increases. When dV/dP is computed by using the difference between the pressure in an open and that in an absolute manometer a greater variation is found. First, this may be observed by referring to table 3 of the experiment on models, in which with increasing pressure not only is the dV/dP quotient not the same as that which is obtained by directly dividing the fluid in the deformation by the actual pressure but, in addition, the differences are marked as the pressure becomes high. It is also observed that these differences are influenced by the area of the elastic membrane, when the dV/dP quotients for systems with diameters of 25, 38, 42 and 106 mm. (table 4) are compared. Similar differences are seen in the $\frac{dV}{dP}V$ quotients.

When by the use of relative and absolute pressures the dV/dP quotients for animals in the horizontal position are computed, it is found that when a head of pressure is successively raised, the value for dV/dP varies as the pressure increases, the changes being divisible into three phases (table 5). The same variations are seen when successive amounts of fluid are added.

In an experiment in which the animal was in the horizontal position, the difference in the pressure of the relative and that of the absolute manometer was 3 cm., the fluid in the manometer being 0.243 cc. and dV/dP, 0.081. When jugular compression raised the relative pressure from 9 to 14.4 cm. the difference between the relative and the absolute pressure was 2.1 cm.; the volume of fluid in the manometer, 0.3888 cc., and dV/dP, 0.185. When the relative pressure was raised to 21.2 cm. the absolute pressure was 9.8 cm. higher; the volume in the manometer, 0.572 cc., and dV/dP, 0.0584. Finally, when the relative pressure was raised to 26 cm. the difference in pressure was 28.5 cm.; the volume in the manometer was 0.702 cc., and dV/dP was 0.0246.

The variations noted when dV/dP is measured in this manner are also seen when pressures are obtained in two manometers of different bore. Here the theory is the same, namely, that the difference in the pressures in the two manometers represents the pressure necessary to

push back into the system a volume of fluid equal to the difference in the volumes of the fluids in the two manometers; the differences in dV/dP may be seen in table 6 for models and in table 7 for animals.

When, in a model at a certain pressure, the open manometer had a cross-sectional area of 0.0379 sq. cm., dV/dP was 0.056; for an area of 0.0812 sq. cm. it was 0.0565; for an area of 0.098 sq. cm., 0.0585; for an area of 0.2676 sq. cm., 0.0511; for an area of 0.7 sq. cm., 0.0446, and for an area of 0.946 sq. cm., 0.0473.

TABLE 5.—*Determinations of* $\frac{dV}{dP}$ *with Successive Increases in the Head of Pressure in an Animal in the Horizontal Position, Computed by Using Relative and Absolute Pressures in an Open Manometer*

Elevation of Head of Pressure, Cm.	Relative Pressure, Cm.	Absolute Pressure, Cm.	Volume in Manometer, Cc.	Difference in Pressures of Relative and Absolute Manometers, Cm.	$\frac{dV}{dP}$ for Relative and Absolute Manometers
1.1	0.6	0.85	0.01884	0.25	0.0754
2.0	1.0	1.45	0.03140	0.45	0.0698
3.0	1.5	2.40	0.04716	0.90	0.0530
4.2	2.5	3.10	0.07850	0.60	0.1310
5.0	3.5	3.80	0.10990	0.30	0.3663
6.0	4.3	5.40	0.13502	1.10	0.1230
7.0	4.9	6.70	0.15368	1.80	0.0854
8.0	6.4	7.50	0.20096	1.10	0.1830
9.0	7.0	8.45	0.21980	1.45	0.1520
10.0	7.4	9.40	0.23236	2.00	0.1160
11.0	8.0	10.40	0.25120	2.40	0.1050
12.0	8.2	11.30	0.25748	3.10	0.0831
13.0	9.2	12.70	0.28888	3.50	0.0803
14.0	10.1	14.10	0.31714	4.00	0.0793
15.0	10.1	14.80	0.31714	4.70	0.0675
15.8	10.6	15.80	0.33284	5.20	0.0640
16.7	10.8	16.70	0.33912	5.90	0.0575
17.7	11.3	17.70	0.35482	6.40	0.0554
18.5	11.4	18.50	0.35496	7.10	0.0500
20.7	11.9	20.70	0.37366	8.80	0.0424

As evidence that these findings are not peculiar to our experiments, we refer to the tables of Weed and his co-workers.[7]

Manometer Bore, Mm.	dV/dP
1	0.154
4	0.131
8	0.146
26	0.179

With two manometers, one with a bore of 1 mm. and the other with a bore of 4 mm., the dV/dP was 0.125; with 1 and 8 mm., 0.145; with 1 and 26 mm., 0.18; with 4 and 8 mm., 0.166; with 4 and 26 mm., 0.207, and with 8 and 26 mm., 0.31.

Comment.—The careful measurements of Weed and his associates have emphasized the importance of elasticity as a property of some of the contents of the craniospinal space and possibly of its enveloping membranes, but we do not believe that they permit one to measure elasticity.

The changes in volume are related to the change in capacity of the elastic membranes, and although in certain known geometric forms the area may be computed from this volume, this is impossible in the

TABLE 6.—*Determinations of* $\frac{dV}{dP}$ *Measured by Using Two Manometers of Unequal Bore, with Successive Increases in the Head Pressure in the Containers*

Head of Pressure	25 Mm. Membrane						38 Mm. Membrane					
	Relative Pressure in Small Manometer,* Cm.	Volume in Manometer, Cc.	Relative Pressure in Large Manometer,† Cm.	Volume in Manometer, Cc.	$\frac{dV}{dP}$ for Two Manometers	$\frac{dP}{dV}V$	Relative Pressure in Small Manometer,* Cm.	Volume in Manometer, Cc.	Relative Pressure in Large Manometer,† Cm.	Volume in Manometer, Cc.	$\frac{dV}{dP}$ for Two Manometers	$\frac{dP}{dV}V$
5	....	.....	....	....	.....	.....	4.20	0.1034	0.75	0.66	0.1580	823
10	3.70	0.0999	0.30	0.24	0.0415	3,133	8.95	0.2416	1.50	1.30	0.1420	921
15	....	.....	....	....	.....	.....	12.00	0.3240	2.20	1.90	0.1600	822
20	7.75	0.2093	0.50	0.44	0.0332	3,922	16.30	0.4401	2.90	2.50	0.1530	865
25	....	.....	....	....	.....	.....	19.90	0.5373	3.50	2.98	0.1490	892
30	12.10	0.3276	0.90	0.67	0.0306	4,262	23.85	0.6439	4.10	3.52	0.1456	917
35	.....	.....	....	....	.....	.....	27.70	0.7479	4.65	4.10	0.1454	924
40	14.90	0.4013	1.10	0.80	0.0290	4,507	31.90	0.8613	5.20	4.48	0.1355	995
45	.....	.....	....	....	.....	.....	35.80	0.9666	5.80	5.00	0.1344	1,008
50	17.90	0.4833	1.25	1.0	0.0310	4,223	40.30	1.0881	6.50	5.60	0.1330	1,024
55	.....	.....	....	....	.....	.....	44.60	1.2042	7.00	6.12	0.1307	1,060
60	21.40	0.5778	1.40	1.18	0.0301	4,355	48.80	1.3176	7.80	6.80	0.1330	1,034
65	.....	.....	....	....	.....	.....	54.30	1.4661	8.45	7.46	0.1300	1,064
70	23.90	0.6453	1.55	1.32	0.0302	4,347	59.70	1.6119	9.40	8.22	0.1310	1,062
75	.....	.....	....	....	.....	.....	65.30	1.7631	10.55	9.30	0.1370	1,024
80	.....	.....	....	....	.....	.....	72.90	1.9683	12.30	10.90	0.1300	1,084
85	.....	.....	....	....	.....	.....	80.60	2.1762	15.60	13.90	0.1800	797

* The bore of the small manometer is 0.027 sq. cm.

† The bore of the large manometer is 0.88 sq.cm.

case of the craniospinal contents. Unless the changes in the area and thickness of the elastic elements are known, it is impossible to measure their elasticity. Weed has pointed out that the quotient dV/dP is not related to a coefficient of elasticity, such as is expressed by Young's Modulus, and we presume he means any other modulus or coefficient. We find that relatively similar quotients so computed can be obtained only when by a constant procedure such as tilting, similar changes in pressure are produced in animals in which at this particular pressure the areas as well as the elasticity of the membranes, etc., are similar. With different pressures produced by different procedures the quotients are entirely dissimilar and are empirical figures measuring nothing.

MANOMETRY

The cerebrospinal fluid pressure is usually measured by a manometer of a type which permits atmospheric pressure to act on the subdural

TABLE 7.—*Variations of* $\frac{dV}{dP}$ *with Different Amounts of Cerebrospinal Fluid Pressure in Animals, Using Two Manometers of Unequal Bore*

Relative Pressure in Small Manometer, Cm.	Volume in Manometer, Cc.	Relative Pressure in Large Manometer, Cm.	Volume in Manometer, Cc.	dV for the Two Manometers	dP for the Two Manometers	$\frac{dV}{dP}$ for Two Manometers
4.4	0.13816	1.20	1.10	0.96184	3.20	0.301
5.0	0.15700	1.10	1.00	0.84300	3.90	0.216
5.3	0.16642	1.30	1.20	1.03360	4.00	0.258
9.0	0.28260	1.30	1.20	0.91740	7.70	0.119
9.5	0.29830	1.90	1.65	1.35170	7.60	0.180
10.8	0.33912	2.00	1.80	1.46100	8.80	0.167
11.5	0.36110	1.98	1.77	1.40890	9.52	0.148
14.5	0.45530	2.10	1.90	1.44500	12.40	0.116
16.8	0.52752	1.90	1.70	1.17200	14.90	0.078
18.5	0.58090	1.80	1.60	1.01910	16.70	0.061

contents. In some cases the pressure is determined in an open tube by reading the height to which the fluid is allowed to flow into the tube on connecting the manometer with a needle which has been inserted into the subdural space. As some fluid has escaped into the manometer, the reading is not that of the absolute or real pressure of the cerebrospinal fluid before the escape of fluid but a relative pressure. The same may be said of aneroid manometers, in which, although the amount of fluid displaced is much less than that in open manometers, some fluid, nevertheless, escapes.

A reading of the absolute pressure may be obtained by using the so-called bubble manometer, in which in a glass tube interposed between the manometer and the spinal fluid a bubble of air has been placed. When the manometer is opened to the cerebrospinal fluid pressure, the bubble is displaced outward. Then the manometer is raised until the bubble has been returned to its original position, and, no fluid having been displaced outward, the absolute pressure is measured by the height

of the column of fluid in the manometer above the level of the needle which had been inserted into the subdural space. Kilgore's [9] apparatus uses this principle by substituting a rubber membrane for a bubble.

Another method for obtaining the absolute pressure is to allow the cerebrospinal fluid to escape into the manometer and then, by elevating the manometer, to push the fluid back into the subdural space until the original level at zero, or atmosphere, is reached. The height of that point above the needle is the absolute pressure. This method has been used clinically by Kausch [10] and Cassidy and Page.[11] We have used it in our experimental studies.

It has been said that if one uses a manometer of small bore, e. g., 1 mm. in diameter, the relative pressure will approximate the absolute

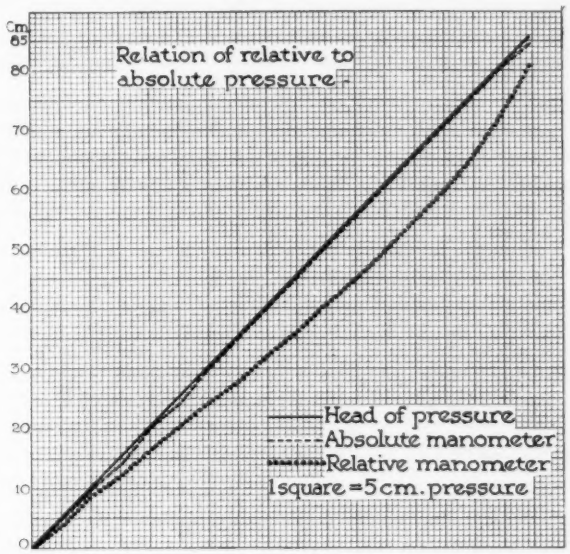

Fig. 8.—Graph showing the relation of relative to absolute pressures in a model with increasing heads of pressure.

pressure obtained by a bubble manometer sufficiently to permit its use in studying the changes in pressure of cerebrospinal fluid. Although this may be true in certain conditions, we have found such large differences under other circumstances that we thought it advisable to study

9. Kilgore, E. S.: Cerebrospinal Fluid Pressure, J. A. M. A. **89**:1856 (Nov. 26) 1927.

10. Kausch, W.: Ein Instrument zur lumbalen Punktion, Injection, und Drucksmessung mit ein Verfahren der letzteren, Deutsche med. Wchnschr. **2**:2217, 1908.

11. Cassidy, M. A., and Page, C. M.: A Method for Determining the Absolute Pressure in the Cerebrospinal Fluid, Proc. Roy. Soc. Med. **4**:56, 1911.

the causes of the larger variations. Kilgore called attention to the inaccuracy of the relative pressure readings in human beings suffering from certain conditions producing high cerebrospinal fluid pressure.

We called attention to the fact that when a cylinder one end of which is closed by a rubber membrane is filled with water and increasing heads of pressure are applied the difference between the relative and the absolute pressure increases as the pressure increases up to a certain point, which we believe to be the proportionality point of the elasticity of the membrane; then it again diminishes. This can be readily visualized in a graph (fig. 8).

We have made similar observations in animals in which we produced changes in cerebrospinal fluid pressure by tilting, compression of the jugular veins and introduction and removal of fluid from the subdural

TABLE 8.—*Comparison of Relative and Absolute Pressures in Animals at Different Degrees of Cerebrospinal Fluid Pressure*

Head of Pressure, Cm.	Average Difference Between Relative and Absolute Pressure, Cm.	No. of Animals
A. Produced by Tilting and Injection of Physiologic Solution of Sodium Chloride		
5-10	1.60	2
10-15	2.42	5
15-20	3.97	10
20-25	6.10	7
25-30	9.70	3
30-35	11.10	6
35-45	19.05	7
45-60	25.50	2
B. Produced by Compression of the Jugular Veins		
5-10	1.50	1
10-15	2.90	3
15-20	4.80	8
20-25	6.10	6
25-30	8.90	8
30-35	9.80	6
35-45	18.60	2
45-55	27.20	2

cisternal space. It may be seen from table 8 that, whatever method of raising the cerebrospinal fluid pressure was employed, the higher the absolute pressure the greater in general was the variation between the relative and the absolute pressure. Of course, some very high pressures exceed those produced by the physiologic processes of the body. Exceptions to this general rule are found in various animals, owing to other factors, which will be dealt with later. The difference between the relative pressure and the height of a column of fluid may be partly understood when one studies a model consisting of a cylinder in which a segment at the top is replaced by a rubber tube and which is filled with water under 10 cm. of pressure and tilted from a horizontal to a vertical position, the rubber tube being uppermost (fig. 9). It can be seen from the graph (fig. 10) that the relative pressure is less than the absolute pressure at the beginning of the curve and is above the

level to which the cylinder is tilted. Then, as the cylinder is raised, centimeter by centimeter, the relative pressure is seen to come closer to the height to which the cylinder is raised, and at a certain point there is a crossing of these lines, after which a divergence again follows. In the meantime, the relative pressure again becomes more and more separated from the absolute pressure.

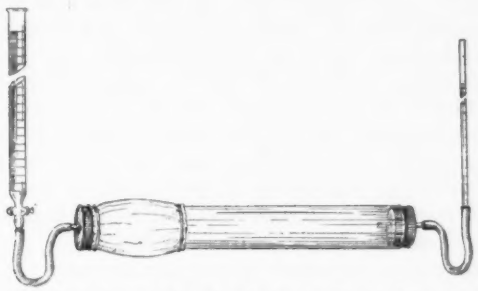

Fig. 9.—Model for study of relative and absolute pressures.

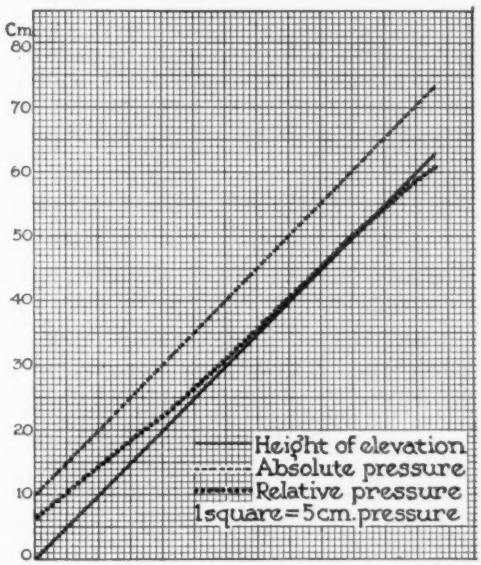

Fig. 10.—Graph showing the relation of relative to absolute pressures when an increase of pressure is obtained by tilting the model illustrated in figure 9.

The explanation of this phenomenon rests on the following considerations:

When pressure is put on an elastic membrane by a head of water it produces a thrust of the membrane, which deformation allows the inflow of an amount of fluid constant for each pressure. Against this thrust the membrane tends to react, and if it is opened to atmosphere an amount of fluid will be expelled cor-

responding to the volume of the deformation. If this fluid is allowed to run into an open manometer only a part of this amount will escape, as the increasing pressure produced by the rising level in the manometer tends to hold back some of the fluid in the membrane. But if the cylinder is tilted so that an additional atmospheric pressure is permitted to act against the fluid in the chamber as it escapes from the chamber, a level in the manometer will be reached at which all the fluid which filled the deformation of the membrane will have escaped. At this point the membrane is at the position at which it was acted on equally from both sides at atmosphere and is acted on equally by the height of the column in the manometer and the height of the column in the cylinder, so that it is not deformed. This is equivalent to saying that the columns in the manometer and in the cylinder have been balanced. Since some fluid has run into the manometer the original conditions have been changed, and if then the amount of fluid in the manometer is forced back into the cylinder by raising the manometer, the pressure will be found to be 10 cm. higher. As the cylinder is elevated above this point the amount of fluid which runs into the manometer will be related to the amount of deformation (then inward) which occurs in this particular membrane when it is exposed to certain pressures. In other words, the amount of fluid which escapes into the manometer will be found to be less than the actual head of pressure imposed by the elevation of the cylinder, by an amount determined by its elasticity and area. This is readily seen when one compares the relative pressures in cylinders of different diameters, one end of each being covered by a rubber membrane of the same degree of elasticity, at the same head of pressure as that of the others. For example, with a head of pressure of 30 cm. of water the relative pressure in the cylinder with a membrane of 25 mm. diameter was 12.1 cm. of water; in one of 42 mm., 23.8 cm., and in one of 106 mm., 30 cm.

That our reasoning is sound in regard to the relation of relative pressure to the height to which a container is raised is shown by the ability to predict the level at which these points will meet. If in a model as described a pressure of 15 cm. of water produces a deformity of the membrane permitting 0.62 cc. of water to run into the cylinder, the area of a cross-section of the manometer being 0.034 sq. cm., it would require a height of 18.2 cm. in this manometer to hold the fluid displaced into the deformation, which would be the point at which the two levels should cross. The actual readings were as follows:

Height of Cylinder, Cm.	Relative Pressure, Cm. of Water
0	2.0
2	3.6
4	5.4
6	7.2
8	9.4
10	11.2
12	12.7
14	14.55
16	16.1
17	17.1
18	18.2
Crossing	
19	18.95
24	23.40

There is another possible reason for the wide variance between the relative and the absolute pressure.

A model (fig. 11) is constructed in which a tambour covered by a rubber membrane and filled with water is placed inside a cylinder filled with water. Over the tambour membrane fits a rigid disk, perforated throughout. This limits the distention of this membrane.

When, then, a head of pressure is put on the membrane a point will be reached beyond which the membrane distends or bulges no farther, because of the disk. Beyond this point relative and absolute pressures are no longer related. If one connects a manometer with the cylinder only a certain amount of fluid escapes into the manometer, whatever may be the pressure, but if one raises the manometer to push back the fluid being pressed against the membrane, the pressure necessary will be that of the head of pressure exerted in the tambour.

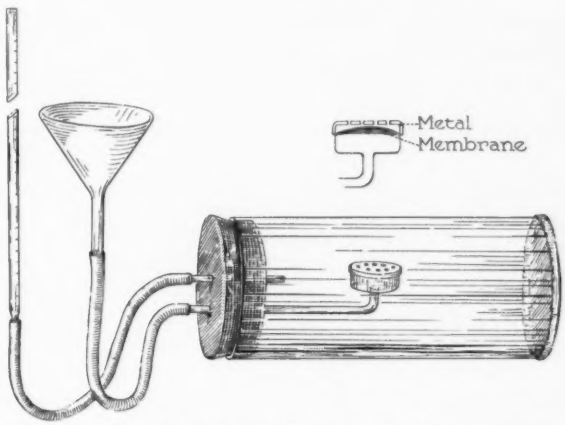

Fig. 11.—Model for study of the limit of distensibility.

In this connection, it is of interest to note the results of the following experiment:

A dog was tilted head down, and fluid was allowed to flow out through a cistern puncture needle until no more escaped at atmospheric pressure. Then 0.5 cc. of fluid was injected, and the relative pressure became 2.1 cm. and the absolute pressure 18.2 cm. With succeeding injections of 0.5 cc. of fluid at a time the readings were:

Relative Pressure, Cm.	Absolute Pressure, Cm.
2.1	18.2
2.7	20.2
3.5	21.9
4.4	23.1
7.2	44.0

When in an intact animal we had an absolute pressure of less than 15 cm. the relative and the absolute pressure under normal conditions of tilting closely approximated each other. Some new factor, producing

this wide variance, has been introduced. Speculation concerning it would be futile. The figures, however, are significant in relation to our conclusion.

Comment.—As a result of these considerations, it is our belief that if accurate studies of cerebrospinal fluid pressures are to be made relative pressures are of no value and only absolute pressures can be used.

MANOMETRY BY DOUBLE PUNCTURES

Deductions have been made from observations of the changes in fluid levels of two manometers, one connected with a lumbar puncture needle, the other with a cistern puncture needle.

Weed [12] stated:

> In some animals, the pressure of the cerebrospinal fluid was taken by occipito-atlantoid and lumbar manometers. Under these conditions, during the control periods when the animal was in the horizontal position, the occipital and lumbar pressures were at the same levels. When, however, the animal was tilted from the horizontal to the vertical, head-down position, the increase in the occipital pressure was far greater than when the dural tube was pierced by only one needle; negative pressures occurred in the lumbar manometer. In one animal, the occipital pressure increased from 120 to 240 mm., while the lumbar pressure fell to minus 100 mm. With the animal in the vertical position, the occipital and lumbar manometers were in the same vertical plane, side by side. The fluid in the two manometers could be seen under these conditions to be exactly at the same level, though in the experimental set-up the lumbar manometer recorded a negative pressure, while the occipital instrument recorded a positive pressure. When the animal was restored to the horizontal position, both these manometers showed resumption of normal pressure.

In order better to understand the relations of the manometers, we constructed a model (fig. 12).

This consisted of a cylinder closed at one end and with the other end covered with a rubber membrane (representing the spinal canal); manometers were attached to parts representing the lumbar and cisternal region. The apparatus was filled with water when in the horizontal position; zero, or atmosphere, was marked on the manometer opposite the level of entrance to the cylinder, and fluid was introduced to this level.

We were surprised to find that on tilting the negative pressures in the lumbar manometer were in no way similar to the figures for pressures submitted by Weed as occurring in his animal when turned head down. In our experiment the negative pressure in the lumbar manometer never read lower than —3 cm. of water. When the relative pressure rose to +41 cm. in the cistern manometer it fell only to —3 cm. in the lumbar manometer. Of course, when the cylinder was tilted about an axis at the "cistern," the zero point of the lumbar manometer was brought opposite the level of the part of the cylinder corresponding to the spinal puncture. If this was not done we noted that when the cylinder was tilted corresponding to the head-down position, the manometers remaining in the positions

12. Weed, Lewis H.: Some Limitations of the Monro-Kellie Hypothesis, Arch. Surg. **18**:1049 (April, pt. 2) 1929.

they occupied when the cylinder was horizontal, the fluid rose to a level of 35.5 cm. in both manometers. If one interpreted this to mean that the pressure rose 35.5 cm. in the cistern manometer and the distance between the level of fluid in the lumbar manometer and the entrance into the cylinder was computed, one might imagine that the negative pressure at this point was — 16 cm. of water. When, as is obviously necessary, the zero point of the manometer was brought to the level of its entrance into the cylinder, the pressure at the "cistern" was 41.5 cm. and at the "lumbar" portion — 1.5 cm., and instead of fluid running into the cylinder it escaped into the cistern manometer. Of course, one is not measuring pressures at all but producing them by balancing two columns of water acting against different levels of the contents of the cylinder.

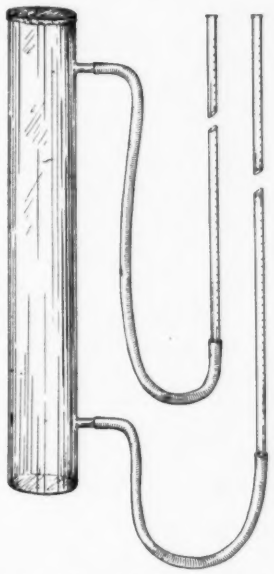

Fig. 12.—Model for study of double punctures.

When we examined a number of animals by similar methods, our results were dissimilar to those of Weed. A rather wide variation was found.

In one animal the cistern pressure rose 16.5 cm., to a level of 20.5 cm., and the lumbar pressure fell 7.5 cm., to a level of — 3.5 cm. In another the pressure rose 21 cm., to a level of 34.5 cm., at the cistern region and fell 13 cm., to a level of — 0.5, at the lumbar region. In another animal it rose 23 cm., to a level of 34.5 cm., in the cistern manometer and fell 9.5 cm., to a level of — 2 cm., in the lumbar manometer. In still another animal it rose 13.5 cm., to a level of 23.5 cm., at the cistern region and fell 14 cm., to a level of — 4 cm., at the lumbar region. In one animal a pressure of — 12 cm. was produced in the lumbar manometer, and a fall of 18 cm. and a rise of 17 cm. occurred at the cistern manometer, producing a pressure of 23 cm. The change was usually greater in the cistern manometer,

indicating that fluid ran out of, not into, the spinal canal. When the change was greater in the lumbar manometer it did not exceed 2 cm., which would represent an inflow of 0.068 cc. of water.

A similar variability of change can be reproduced in a model (fig. 13). The contents of a cylinder similar to the one described in figure 12 are acted on by a membrane subjected to a head of pressure. By increasing the pressure a state may be brought about in which the system acts as a rigid one closed at the top and bottom, under which condition the changes in the "cistern" and "lumbar" manometers are the same when the model is tilted.

Comment.—Our studies do not bear out the observation that there is a considerable displacement of fluid into the subdural space when two

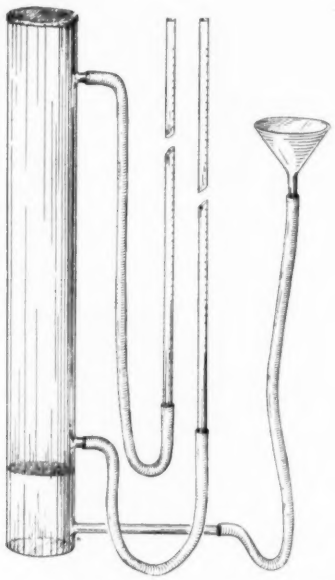

Fig. 13.—Model for study of double punctures.

manometers act against each other, provided that the zero points of the manometers are placed opposite their respective entrances into the subdural space.

The only factor which we found could be studied by this method is the pressure necessary to push back a certain amount of fluid from a manometer into the subdural space by changing the relationships of another manometer connected with that space.

THE RIGID CONTAINER

If the cerebrospinal fluid were exposed to full atmospheric pressure at all times and in all positions assumed by man or imposed on animals, the changes in pressure would be related to the laws governing a simple

hydrostatic column and could easily be understood. This, however, is not the case. Weed [13] has stated: "All of the conceptions of the mechanism for the maintenance of this [cerebrospinal fluid] pressure are based primarily upon the rigid character of the bony coverings of the nervous system." Since the concept that the cerebrospinal axis is situated in a "closed box" was advanced by Monro [14] in 1783, developed by Kellie [15] and modified by Burrows,[16] this hypothesis has entered into the consideration not only of the maintenance and changes of cerebrospinal fluid pressure but of the intracranial volume of blood and cerebrospinal fluid, the pulsation of the vessels, the strain on and circulation through them and many other functions.

The terms closed box and rigid container have led to certain faults in reasoning, in an attempt to prove the validity of the Monro-Kellie hypothesis. It is perhaps needless to point out that the rigid skull is open at the bottom both to the spinal canal and to the blood vessels. If the intracranial contents were actually encased in a rigid and closed box, negative pressures could not be produced by differences in osmotic tensions in the intravascular and extravascular elements. This would be analogous to a change in pressure as a result of osmosis in a rigid container divided into two parts by an animal membrane, one part containing a physiologic and the other a hypertonic solution of sodium chloride. On the other hand, in an attempt to show that the spinal dural tube does not serve as an absolutely rigid container, we find such a statement: "If the tube were a truly rigid container the tiltings from the horizontal to the vertical positions, either tail-down or head-down, should not in any way affect the pressure of the occipital cerebrospinal fluid, the tube being to all intents completely filled with fluid." This seems to imply that changes in pressure are dependent on movement of the fluid in containers and not on its weight. It should be needless to point out that if one came to such a conclusion because one could not observe a change in an open manometer when such a tube was tilted, one should not be surprised if, when using a tube longer than about 34 feet (11.23 meters), a change in pressure is observed and should recall that "nature abhors a vacuum" but that "nature's horror of a vacuum does not extend beyond 32 feet (Galileo)."

13. Weed, Lewis H.: The Cerebrospinal Fluid, Physiol. Rev. **2**:171, 1922.

14. Monro, Alexander: Observations on the Structure and Functions of the Nervous System, Edinburgh, Hamilton, Balfour & Neill, 1783.

15. Kellie, George: Appearances Observed in the Dissection of Two Individuals: Death from Cold and Congestion of the Brain, Tr. Med.-Chir. Soc. Edinburgh **1**:83, 1824.

16. Burrows, George: On Disorders of the Cerebral Circulation, London, Longman and others, 1846.

Recently Weed and Flexner [17] approximated the truth when they said:

> It is realized in the intact animal on such abrupt tilting from the horizontal to the two vertical positions pressure changes occur in the cerebrospinal fluid in the occipital region whether the spinal tube be completely rigid or not, but in such a system with an open-end manometer of small bore, dislocation of fluid is essential for the recording of any change in pressure.

The same changes in pressure occur in a column of water within a rigid container as in a column open to atmosphere, but they must be measured by different methods.

The development of a negative pressure at, or near, the top of a container does not mean that the top of the container is absolutely rigid. When a cylinder closed at its top and open at the bottom is filled with water and the bottom rests in a basin filled with water, thus exposing the bottom to full atmospheric pressure, or about 34 feet (1,122 cm.) of water, the pressure at the top is an amount less than 34 feet (1,122 cm.) by the length of the cylinder, or less than atmospheric pressure by a length of the cylinder. If the top of the cylinder is closed by an elastic membrane the negative pressure will be diminished by an amount related to the part of full atmospheric pressure acting through the membrane on the column of water, but the pressure will still be negative.

Monro himself indicated that the contents of the skull are open to blood vessels. Arnott,[18] a good physisicist of his day, stated:

> The head may be considered as an airtight vessel or cavity of bone, containing chiefly brain and blood, and having openings which admit blood vessels leading to and from the heart. The atmospheric pressure, therefore, always keeps the head full, as it keeps the top of a siphon full.

It should be pointed out that it is not the rigid character of the skull which keeps it full but atmospheric pressure acting against the contents of this container, which is open at the bottom.

The skull is also open at the bottom to the spinal canal. How much atmospheric pressure acts from without the bony covering of the canal and other points of action will be left for a later section of the paper. It can be seen from the diagrammatic representation of conditions existing in a skull open to blood vessels and the spinal canal that they are quite different from those in a closed box. In such a model (fig. 14) positive and negative pressures can be produced at the cistern by appropriate tilting, and this would hold true even if no atmospheric pressure acted on the contents of the spinal canal from without.

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17. Weed, Lewis H., and Flexner, Louis B.: Further Observations upon the Monro-Kellie Hypothesis, Bull. Johns Hopkins Hosp. **58**:196, 1932.

18. Arnott, Neil: Elements of Physics or Natural Philosophy, London, Longman, Rees, Orme, Brown & Green, 1828.

The concept of the closed box or rigid container has led to statements which should be restudied. Howe [19] stated:

> Theoretically, there might be a reciprocal variation in the volumes of blood and cerebrospinal fluid within the dural cavity. Increase in the amount of cerebrospinal fluid might be compensated for by an equal diminution in the blood of the cerebral circulation. It has been shown, however, in the preceding discussion that this is not the case, at any rate when the volume of increment of cerebrospinal fluid is not large. Were there a decrease in blood volume equal to the accession of cerebrospinal fluid, there would be obviously no increase in intracranial tension and a distortion of the nerve tissue due to the alterations in special relationship would result.

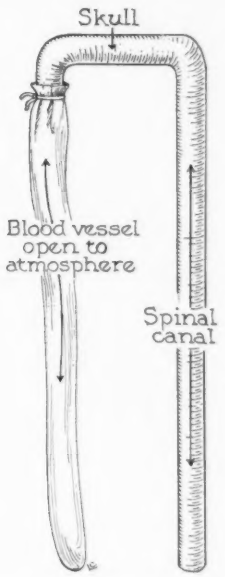

Fig. 14.—Diagrammatic representation of a skull open to blood vessels and the spinal canal.

We did not find this to be true when we constructed a model (fig. 15) in which a constant head of pressure of water distended a membrane covering a tambour, which was enclosed in a rigid cylinder filled with water. The head of pressure distended the membrane a certain amount, *A*. When the fluid was introduced into the cylinder the membrane was thrust into an opposite direction, to *B*. It can readily be seen that if the manometer is opened the membrane will first contract to its neutral position and then will be thrust outward by the head of pressure and that the height to which the manometer must be raised to thrust the

19. Howe, Hubert S.: Physiological Mechanism for the Maintenance of Intracranial Pressure, A. Research Nerv. & Ment. Dis., Proc. **8**:7, 1929.

membrane back to the position *B* will be greater than that necessary to thrust it back to its neutral position, 0. When a pressure is exerted on a container of fluid through an intermediate membrane changes in pressure result from the introduction or removal of fluid, despite the constancy of the volume of the container.

Fremont-Smith and Kubie [20] alluded to the same argument as did Howe. As a further proof of the independent contractility of the intracranial vessels, in the existence of which we believe, they said:

> In the first place, it would be impossible to produce a negative intracranial pressure at all, if the intracranial arteries did not possess a powerful contractile tone: for in the absence of such arterial tone, every effort to reduce intracranial pressure would be accompanied by a corresponding increase in volume of intracranial blood, and as the effort was pushed further, the animal would gradually bleed into his cranial vessels—in the same sense in which it is said an animal in shock bleeds into its splanchnic vessels.

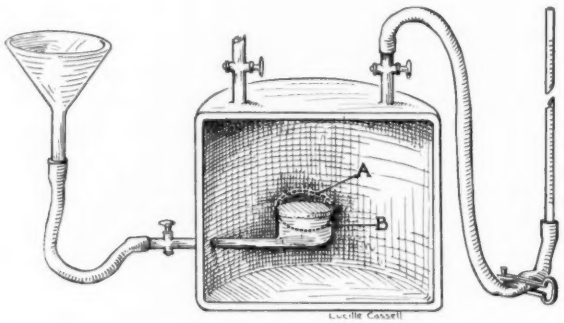

Fig. 15.—Model to demonstrate changes in pressure despite reciprocal changes in volume.

In regard to the first part of their statement, the same forces are at work when one removes fluid surrounding a membranous system as when one introduces fluid into it. When negative pressures are produced by tilting to a tail-down position there is a state analogous to that in the model (fig. 16), in which a balloon filled with water under a constant head of pressure is placed in a chamber closed at the top and open at the bottom to a tube, the bottom of which is open to a funnel covered with a rubber dam, the system being filled with water. The contents of the cranial, or upper, part of the container are constant, whether the funnel is raised or lowered; as the amount of fluid outside the balloon increases that within diminishes, and the pressure falls when

20. Fremont-Smith, Frank, and Kubie, Lawrence S.: The Relation of Vascular Hydrostatic Pressure and Osmotic Pressure to the Cerebrospinal Fuid, A. Research Nerv. & Ment. Dis., Proc. **8**:104, 1929.

the funnel is lowered and rises when it is elevated. If the funnel is lowered sufficiently, a negative pressure is produced at the place of junction of the "cranial" and "spinal" portions.

STRAIN ON VESSELS

The latter part of the statement of Fremont-Smith and Kubie introduces the question of strain on the intracranial vessels. Grashey [21] pointed out the physical laws governing strain on vessels traversing a rigid container. The results of the strain are related not alone to the contractility of such a vessel but to its distensibility, elasticity and other properties, placing a limit on the amount of distortion which can be produced.

Since, as will be shown, when atmospheric pressure is excluded from the contents of the blood vessels of the craniospinal contents by occluding the entrances and exits of these vessels, enormous negative pressures are necessary to remove a drop of cerebrospinal fluid, it follows that there must be a reciprocal dislocation of the vascular bed when cerebrospinal fluid is removed.

When a vessel or tube traverses a container which is closed at the top and open at the bottom to atmosphere through a rubber membrane and is filled with water, the strains within and without the vessel are equal when in the horizontal position. When the container is tilted with the membrane down a negative pressure is produced, and an outward strain occurs in the vessel. If it were true that only these factors are at work when negative pressures are produced by tilting "tail down," complete distention of the vessel might occur, but another factor must be considered in relation to the forces at work in the cerebrospinal container in animals and man. In addition to the opening into the spinal canal, the cranial contents are also open to the arteries and veins. When an animal is tilted tail down, there is a diminished pressure not only in the spinal fluid at the top of the column but in the vascular bed of the cranium as well. The state is analogous to that in a model in which a vessel under a constant head of pressure traverses a cylinder filled with water, one end of the cylinder being closed by a rubber membrane and the end of the vessel closed by a rubber membrane outside of the cylinder (fig. 17). If the lengths of the cylinder and vessel are equal, the outward and the inward strain remain equal.

The principle may further be illustrated by constructing a model (fig. 18) in which at a constant head of pressure fluid is made to run

21. Grashey, H.: Experimentelle Beiträge zur Lehre von der Blut-Circulation in der Schädel-Rückgratshöhle, in Festschrift der med. Fakultät der Universität München zur Feier des fünfzigjährigen Doctor Jubiläums des Professor Ludwig Andreas Buchner, München, J. F. Lehmann, 1892.

through a balloon within a rigid container filled with water and open at the bottom to a funnel covered by a rubber membrane. The vessel through which fluid escapes from the balloon has its outlet at the level of the funnel and the two may be raised and lowered simultaneously. Although negative pressures are developed within the container, the balloon distends only imperceptibly, and as the outlets of the funnel and vessel are lowered together fluid escapes more rapidly from the vessel. If the outlet to the vessel is occluded continued distention of the balloon occurs when the funnel is lowered.

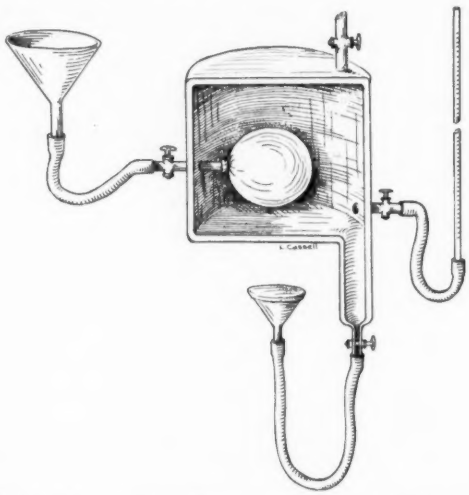

Fig. 16.—Model to demonstrate changes in pressure despite reciprocal changes in volume.

CEREBRAL CIRCULATION

The relation of negative pressures to the circulation of the blood has led to many fancies. Many years ago it was thought that the return of blood from the veins of the lower extremities was the result of negative pressure developed in part of the heart. Even recently, because it was imagined that the distance from the heart to the brain of a giraffe is so great that the pressure at the arch of the aorta is insufficient alone to force the blood into the brain, it was thought that the negative intracranial pressure might have some function in bringing about adequate circulation.

Arnott [18] in 1828 pointed out some of the fallacies relating to these theories. "Since atmosphere acts upon unit areas, the 'suction' of blood upward in a vessel would produce a collapse of a vessel when but a few inches have been moved." Although it was unnecessary, we repeated the experiment, as a matter of interest.

When a straight glass tube was connected to the carotid artery of a dog, the column of blood rose about 8 feet (243 cm.). If by means of a syringe an attempt was made to suck the column of blood upward, it could be raised an additional 5 inches (12.7 cm.) when the artery was collapsed and occluded.

The only manner in which a rigid box open at the bottom can affect circulation within it is by acting as the top of a siphon. The increased flow into the box, however, occurs only during the period of change in hydrostatic conditions, as in tilting. Once the tilting is concluded and the system again at rest, no further increased flow occurs. This can be illustrated by the model described as figure 16, in which the balloon distended during tilting and came to rest at the termination of the tilting.

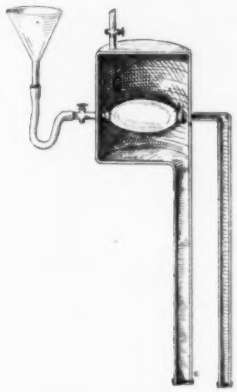

Fig. 17.—Model for study of strain on tubes traversing a container.

FUNCTION OF ELASTICITY

Recent experiments led Weed [22] to a conception of the central nervous system as surrounded by membranes and vascular channels possessing elasticity. He stated:

The cranial walls and the vertebral arches may be looked upon as affording rigidity to the whole system and as protecting it from atmospheric pressure, but there is within this bony container a complex elastic element, constituted of the spinal dural sac and also of the blood vascular channels within the nervous system and the meninges. This elastic element permits, in the intact animal, a dislocation of fluid from one part of the nervous system to another without appreciable change in volume. When however the volume is altered by external displacement of fluid, the elasticity of the system permits this external dislocation as well as the internal readjustment of the fluid volumes.

22. Weed, Lewis H.: Positional Adjustments of the Pressure of the Cerebrospinal Fluid, Physiol. Rev. **13**:80, 1933.

This interesting conception leads to the question how and under what conditions the elastic property of this complex element can contribute to the external dislocation of fluid.

Many misconceptions arise from confusion of the terms elastic and distensible and inelastic and rigid; bodies may be highly elastic and rigid, as is steel, and distensible and inelastic, as is gum. Elasticity is the property of matter by which it returns to its original state when, it having been deformed by a force, the force is removed.

The tension of an elastic membrane, such as that of a balloon, is produced by the action of a greater pressure within than without. This may be brought about by exposing its interior to a pressure greater than atmosphere, by introduction of gas or liquid or by reducing the pressure outside it, as by the creation of a partial vacuum, the interior being exposed to atmospheric pressure.

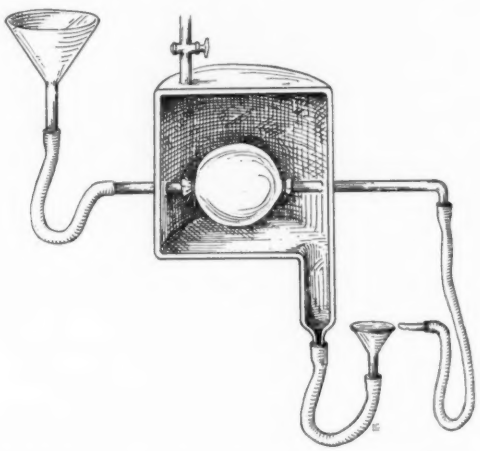

Fig. 18.—Model for study of strain on tubes traversing a container.

The changes in volume in the balloon are the results of changes in the pressure relationship within and without the balloon. The extent of the change in volume is related to the area and elasticity of the balloon. The changes in volume do not produce the changes in pressure, but the changes in pressure produce the changes in volume.

To every action of force in nature there is an equal reaction. Therefore, when the elastic balloon is distended it reacts in an opposite direction, and if the balloon is punctured it contracts to nearly its original position, provided that it possesses a high proportionality point of elasticity. The contents of the balloon would then be displaced outward. But this cannot occur under all conditions. However elastic the balloon may be, the return to its original position when it is punctured after distention is impossible unless atmospheric pressure acts on the

outside of the balloon, except when the balloon is capable of exerting a force of over 15 pounds (6.8 Kg.) to the square inch (6.45 sq. cm.). A balloon may be distended with water and inserted in a rigid box which can be completely closed, the remaining space being filled with water, a condition which is in part comparable to the cerebrospinal fluid enclosed in an elastic complex within a rigid container. If, then, a needle is inserted into the balloon, no measurable amount of water is displaced outward. The pressure outside would have to be reduced almost a full atmosphere before any fluid could be displaced, that is, 34 feet (1,122 cm.) of water less the amount represented by the elastic force of the balloon, which, if comparable to that of biologic tissue, is probably rela-

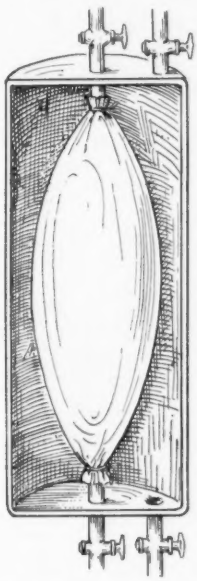

Fig. 19.—Model for study of elasticity of a balloon in a rigid container.

tively small when one remembers that the force of the heart beat propels a column of water only 8 feet (264.2 cm.) in height. If the balloon were cylindric and enclosed in a cylindric box under the same conditions, tilting from the horizontal to the vertical position would not permit dislocation of fluid from within the balloon after puncture, as we found when we, perhaps unnecessarily, made the model illustrated in figure 19. Since the neural axis is open to atmospheric pressure through the blood vessels at least, if the remaining parts of the coverings of the neural axis, both cranial and spinal, are considered to be rigid, one would have a system somewhat similar to the model shown in figure 20. In this model water under a constant head of pressure acting on an elastic membrane, analogous to the blood vessels, is enclosed in a rigid cylinder,

near the end of which is another elastic membrane, representing the meninges, dividing the cylinder into two parts, *A* and *B*, both filled with water. Under such conditions the elasticity of the membrane at *M* plays no part in any dislocation of the fluid when a needle is inserted in the cylinder at *A*, the whole cylinder acting as a closed container. If, however, the end of the cylinder is closed by an elastic instead of a rigid membrane, the head of pressure produces a thrust of the tambour membrane, and the transmitted pressure, a thrust of the membrane *M*, representing the meninges. This membrane reacts against the thrust, and if a needle is introduced through it into part *A* of the cylinder, fluid will be displaced outward in a quantity related to the area and elasticity of both the tambour and the membrane *M*.

It can be seen that only if the meninges are open to atmospheric pressure from without can any property of elasticity play a part in the external dislocation of intradural fluid. The elasticity of the blood vascular channels prevents rather than facilitates the escape of fluid, for the greater their elasticity the less their distention.

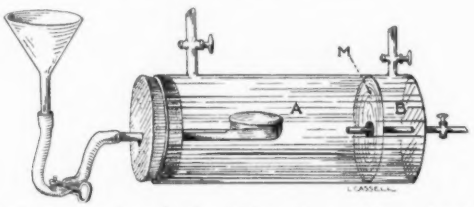

Fig. 20.—Model for study of elasticity of a rubber diaphragm in a rigid container.

RESULTANT OF FORCES

As the result of extensive and careful studies, Weed [23] concluded that changes in pressure in the cerebrospinal fluid are dependent on two clearly defined hydrostatic elements. He stated:

> The first of these, which we have termed the "meningeal factor," relates to the hydrostatic changes in the pressure of the cerebrospinal fluid as modified by the elasticity of the meningeal sac and its method of suspension within the body. The second factor, called "the vascular reflection in the cerebrospinal fluid," is the effect of the hydrostatic pressure-change within the intradural blood vessels as the pressure change becomes modified by the physiological elasticity of the vessels themselves and reflected upon the pressure of the cerebrospinal fluid.

As the result of analysis of the change in pressure when one or the other of these factors was excluded, Flexner and Weed [24] concluded

23. Weed, Lewis H.: Some Aspects and Problems of Intracranial Pressure, Bull. Johns Hopkins Hosp. **52**:345, 1933.

24. Flexner, L. B., and Weed, Lewis A.: Factors Concerned in Positional Alterations of Intracranial Pressure, Am. J. Physiol. **104**:681, 1933.

that the total "effect must be due to the greater of the two columns; under no circumstances could this effect be the addition of the two columns."

More fully to explain their reasoning, we quote extensively their description of one of the experiments:

> In this type of preparation . . . [i. e., with a ligature about the upper part of the spinal cord] the head-down tiltings yielded the same increases in the pressure of the occipital cerebrospinal fluid and of the superior sagittal sinus as did similar positional alterations in the intact animal. The tail-down tiltings, with the whole splanchnic area presenting a reservoir for hydrostatic flow of the blood due to vasomotor paralysis from the ligature, gave pressure-decreases more than double those of intact preparations. With the identity in the head-down pressure-reactions of the cerebrospinal fluid, in the intact dog and in the dog with

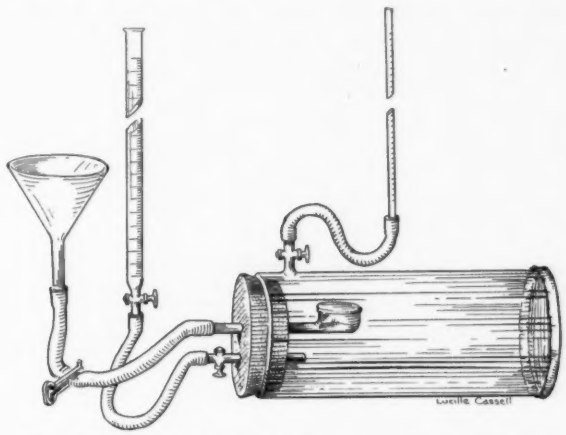

Fig. 21.—Model for study of the resultant of forces.

> ligated cord, one is led inevitably to the interpretation that the pressure-increase in the cerebrospinal fluid on such head-down tiltings is determined by the vascular reflection, and in turn is unaffected by the decrease in the meningeal mechanism through the exclusion of a large part of the spinal subarachnoid space. That the pressure-increase of the cerebrospinal fluid in the dogs with ligated spinal cords should be exactly the same as that of the intact animal on such head-down tilting must be far more than mere coincidence; it can best be taken to mean that the pressure-increase, due to one of the two factors (here the vascular reflection), is alone necessary to account for the pressure-change recorded in the cerebrospinal fluid.

We thought that it would be of interest to study this conclusion.

The conditions present when a vascular reflection and a hydrostatic column are jointly at work may be approximated in a model (fig. 21) in which a tambour covered with a rubber membrane and filled with water at a constant head of pressure is placed within a cylinder, one end of which is closed by a rubber membrane. Into the cylinder a column of water in a buret is introduced, and an open

manometer is applied. The vascular reflection is represented by the thrust of the tambour membrane, resulting from the constant head of pressure. With the hydrostatic column closed we found that a head of pressure of 5 cm. produced a pressure within the cylinder of 1.9 cm.; one of 10 cm., 3.8 cm.; one of 15 cm., 5.4 cm.; one of 20 cm., 6.8 cm.; one of 25 cm., 8 cm., and one of 30 cm., 9.1 cm.

When, then, the hydrostatic column was opened at a pressure of 5 cm., the resulting pressure was 13 cm.; at 10 cm. it was 16.8 cm.; at 15 cm., 20 cm.; at 20 cm., 23.2 cm., and at 25 cm., 26.2 cm.

When the hydrostatic column acted against the outside of the tambour membrane with a pressure of 5 cm., the head of pressure against the inside of the membrane was 30 cm., and the resulting thrust of the membrane was due to the excess of 25 cm. of pressure from within. We had found that a pressure of 25 cm. acting against the tambour produced a pressure of 8 cm. in the cylinder; if we added this to the hydrostatic column of 5 cm. we obtained 13 cm., which is the exact figure we obtained when we measured the absolute pressure with a manometer. With a hydrostatic head of 10 cm. and a head of pressure of 30 cm. a force of 20 cm. acts against the tambour, and, according to our figures, this should produce a pressure of 6.8 cm. in the cylinder; by adding 10 cm., or the height of the hydrostatic column, a total of 16.8 cm. is obtained, and when measured by a manometer the pressure was 16.8 cm. With a column of 15 cm. the estimated pressure was 20.4 cm. and the actual pressure 20 cm.; with a column of 20 cm. the estimated pressure was 23.8 cm. and the actual pressure 23.2 cm., and with a column of 25 cm. the estimated pressure was 26.9 cm. and the actual pressure 26.9 cm.

This remarkably close approximation of the computed and the measured pressures leads us to conclude that when two forces act against a body the total force exerted is the resultant of these forces, irrespective of the preponderance of either—perhaps not an unexpected conclusion.

POINT OF ACTION OF ATMOSPHERE ON THE CEREBROSPINAL FLUID

Monro's [14] concept that the brain is "enclosed in a case of bone" has been extended to include the entire craniovertebral contents. Into this rigid cavity arteries enter, and from it veins depart. At first the concept was based on anatomic generalizations, but later it was supported by indirect experiments on models, animals and man. Such experiments have dealt with the observations that in the erect position the cerebrospinal fluid pressure is less than atmosphere above the level of the foramen magnum, that negative pressures are produced in the skull by the intravenous administration of hypertonic solutions and that the change in the cerebrospinal fluid pressure produced by tilting an animal to the head-down position is greater than when the animal is tilted to the tail-down position.

Grashey,[21] in a study on models, pointed out certain differences in the cranial and spinal cavities. He compared the physical condition to a system in which a vessel with rigid walls (the skull) was immersed in another vessel with rigid walls (the spinal canal) but in which a

membrane (the dura) was surrounded by a filled plexus of tubes (the epidural veins). Since the skull is open to the blood vessels at its base, he reasoned that this is equivalent to a membrane at the point of junction of the skull and the spinal canal and that, therefore, in the erect position the zero point would lie at the boundary of the craniovertebral cavities, at about the level of the foramen magnum. Above this level a negative pressure exists, and below, to the end of the spinal canal, a positive pressure. He expressed the opinion that in the reverse, or head-down, position the entire column from the end of the spinal canal to the vertex would act in a positive direction. He noted the entrance of vessels into the spinal canal at various levels but asserted that, although there would be a general lowering of the zero point, the change would be negligible.

When Krönig and Gauss [25] showed that with the subject in the sitting position a column of fluid in an upright tube had its upper level only in the upper thoracic or lower cervical region, Propping [26] introduced the concept that the spinal epidural space constitutes a wall possessing a certain elasticity. Because of this property, there is displacement of fluid downward in the erect position, and for this reason there is a lowering of the zero point. This displacement, or dislocation, of fluid, presumed to occur because of the dilatation and compression of the intradural vascular bed produced by inward collapse of the spinal dural sac, with stretching of the epidural fibers and dilatation of the epidural venous plexus, was extensively studied by Weed,[22] who likewise studied the elasticity of the elastic elements.

In studying another problem, we had constructed a model (fig. 22) in which a constant head of pressure was transmitted to the contents of a rigid container, in the lower, cylindric part of which was a rubber tube, likewise filled with water, communicating with the upper ("cranial") part, the space around the rubber tube being filled with water. The changes in pressure at the junction of the "cranial" and the "spinal" part on tilting to the "head-down" and "tail-down" positions were the same. We concluded that neither the elasticity of the rubber tube nor its collapsibility played any part in the change of pressure, the system acting as a rigid container, into which a head of pressure was transmitted through the membrane. Under this condition the changes in pressure on "head-down" and "tail-down" tilting would be the same.

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25. Krönig, B., and Gauss, C. J.: Anatomische und physiologische Beobachtungen bei dem ersten Tausend Rückenmarksanästhesien, München. med. Wchnschr. **54**:1969, 1907. Krönig, B.: Anatomische und physikalische Betrachtungen über Rückenmarksanästhesie, Beitr. z. Geburtsh. u. Gynäk. **12**:127, 1909.

26. Propping: Die Mechanik des Liquor cerebrospinalis und ihre Anwendung auf die Lumbalanästhesie, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **19**:441, 1908.

This conclusion led us to study the problem by another method. It is known that when a rigid flask filled with water is inverted and the neck opened to atmosphere by a stopcock no fluid escapes. Since the contents are held in place by full atmosphere, no fluid can escape unless the flask is longer than about 34 feet (11.23 meters). If the interior of the flask is open to atmosphere through a U-shaped tube filled with water the limb of the tube open to atmosphere would have to be lowered about 34 feet before any fluid would escape from the flask. Since, under this condition, the meniscus of the water in the open limb of the U-tube would be stationary, for every centimeter the U-tube is lowered there would be an added centimeter of negative pressure at the top of the flask. If, however, the contents of the flask are open to atmosphere at the top through a rubber membrane and at the neck through a U-tube

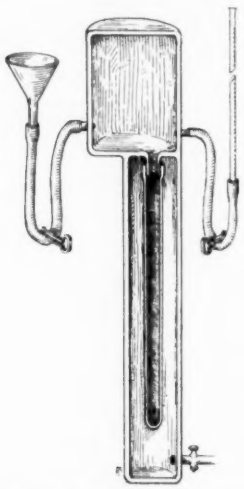

Fig. 22.—Model for study of changes in pressures produced on tilting.

filled with water, a certain amount of fluid escapes from the flask into the tubing when the open limb of the U-tube is lowered centimeter by centimeter, until full distensibility of the rubber membrane at the top is reached and the system again becomes rigid at the top, when no further fluid will escape.

Two burets were connected by pressure tubing and filled with mercury nearly to their tops. One buret, which was calibrated in millimeters and had a cross-sectional area of 0.24 cm., was connected to a needle by pressure tubing filled with physiologic solution of sodium chloride. The position of the meniscus of mercury was marked on the buret, and the meniscus was brought to the level of the cistern into which the needle was inserted. The buret open to atmosphere was lowered by a ratchet-driven mechanism, diminishing the head of pressure centimeter by centimeter, and readings were made.

In three types of preparations of animals measurements were taken to determine at what point fluid escaped from the cerebrospinal subdural space at the cistern when the mercury column was lowered centimeter by centimeter and, likewise, the distance to which the mercury column could be lowered in two of these preparations and fluid continue to escape.

The first preparation was a normal animal, either dead or alive. The cerebrospinal contents were open to atmosphere through the cerebral and spinal vessels. The second preparation was an animal in which the action of atmosphere through the cerebral vessels was excluded by the following procedure: The vessels entering the skull were tied close to their entrance; the rest of the tissue on the undersurface of the skull, including the lower jaw, was dissected away, and the vessels entering the chest were tied. The whole base of the skull was then covered with plaster of paris. Operation was performed on the animals when under ether anesthesia, but they usually died before the termination of the operative procedure, so that a reading of the manometer was possible only a short time after death, usually from ten to fifteen minutes.

In the third preparation, in addition to tying the cerebral vessels and applying plaster of paris to the base of the skull, the animal was eviscerated; many vessels destined to enter the spinal canal were tied close to their entrance, and the anterior surface of the spine was covered with plaster of paris.

We had, then, (1) a preparation in which the cerebrospinal contents were open to atmosphere through both cerebral and spinal vessels, (2) one open to atmosphere only through the spinal vessels and (3) one in which atmosphere could not act through any vessels.

When atmosphere could act through both cerebral and spinal vessels, i. e., in a normal animal, the results were the same in the living animal as in an animal observed soon after death. With every drop of 2 cm. in the level of the meniscus of the mercury column there was an escape of fluid into the other limb of the U-manometer of somewhat less than a length of 2 cm. in a column with a cross-sectional area of 0.24 cm., so that when the column of mercury had been lowered 74 cm. a column of fluid of 62.5 cm. had escaped from the cerebrospinal cavity, amounting to 15 cc. This experiment shows that the cerebrospinal contents are open to atmosphere through the cerebrospinal blood vessels and that enormous negative pressures can be applied to them before full distensibility is reached (fig. 23).

In the second preparation, in which atmosphere could act only through the spinal vessels, it was found that for every centimeter the column of mercury was lowered there was an escape of fluid until the mercury bulb had been lowered 14 cm., when no further escape occurred and the preparation acted as a system rigid to atmosphere above. This indicated that atmosphere acted through the spinal vessels alone but that the limit of distensibility of these vessels was reached much earlier than when the combined cerebral and spinal vessels were open, constituting a physical expression of a known anatomic fact.

In the third preparation atmosphere could not act through either cerebral or spinal vessels. The column of mercury was lowered to a level representing 8 feet (264 cm.) of water before any fluid escaped from the craniovertebral cavity, and then only because a small bubble of air entered at the junction of the needle and its connections. From this point only a few millimeters of fluid continued to escape, so that when the column of mercury had been lowered 40 cm., or an equivalent of over 17 feet (561 cm.) of water, a column of only 2.5 cm. of fluid had escaped as air entered.

This seems to us to be crucial proof that atmosphere acts on the cerebrospinal fluid not directly through bony or ligamentous coverings or the like but only through the blood vessels.

Since atmosphere acts on the cerebrospinal fluid through the blood vessels, it seems that the reason for the position of zero, or atmospheric pressure, in the cerebrospinal cavity, which is usually found with the subject in the erect position somewhere below the level of the foramen magnum, must be sought in the degree of atmospheric pressure transmitted through the vessels in that position. It is suggestive that the point of reference for the venous system from the head is somewhere

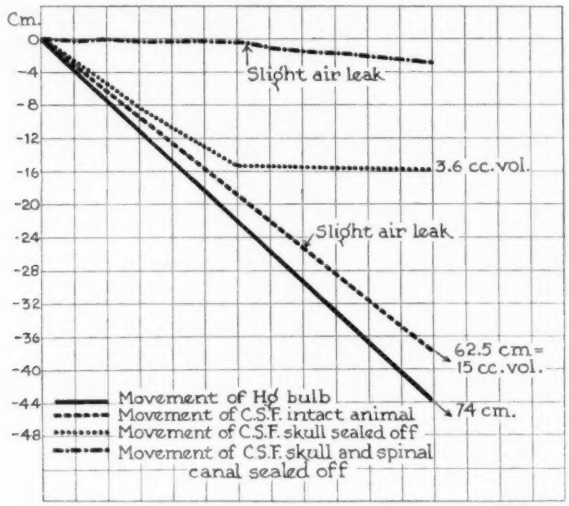

Fig. 23.—Graph illustrating the escape of cerebrospinal fluid when the zero point of atmospheric pressure is successively lowered.

below the clavicle when the subject is in the erect position, and the zero point of the cerebrospinal fluid pressure, in the lower cervical region.

For the same reason, if the change in cerebrospinal fluid pressure on tilting from the horizontal to the head-down position is greater than that resulting from tilting to the feet-down or tail-down position, the explanation must be sought in changes of pressures in the vascular system. According to this theory, it should be found that there is a greater change in the pressure within the vascular system in tilts to the head-down position than in those to the feet-down or the tail-down position.

Weed [22] showed that the pressure in the superior sagittal area of the dog rises an average of 184.1 mm. of water on tilting to the head-down position and decreases an average of 79.7 mm. tilting to the tail-down position.

Henderson and Haggard [27] stated:

> In a man standing at rest or sitting, the veins of the neck are collapsed; the blood aided by gravity runs down from the head so rapidly that the stream has no "surface," no head of pressure" that can be measured. The meniscus, so to speak, of the column of blood in the superior vena cava is somewhere below the upper edge of the clavicle and therefore not visible.

When these authors tilted a man to an angle of 45 degrees in the head-down position the level of the meniscus transected the trunk at about the level of the umbilicus. With the subject in the head-up position the distance from the vertex to the meniscus was much shorter than that in the reverse, or feet-up, position. In other words, the hydrostatic effect should be less in the feet-down than in the head-down position. Suggestive of an explanation of this is the conclusion of Clark, Hooker and Weed,[28] who found that instead of a single point of reference at the level of the right auricle from which the hydrostatic factor in venous pressure is measured, the venous system in the living animal is broken at the heart and must be considered as two columns, each with its own point of reference found to be located a short distance from the heart, that for the tail section 121 mm. from the heart, and that for the head section, 38 mm. distant.

This is in keeping with the fact that, given a single head of pressure acting through a membrane on the contents of a rigid container, the changes in the head-down and the tail-down position are the same. When, however, the head-down and the tail-down position are acted on by different heads of pressure the changes in these respective tilts are different.

Comment.—The cerebrospinal contents are exposed to atmospheric pressure only through the blood vessels.

The reason for the difference in the cerebrospinal fluid pressure in the head-down position and that in the tail-down position is the difference in pressure in the blood vessels in these respective positions.

CONCLUSIONS

Since the area and elasticity of enveloping membranes are related to the ratio between the fluid removed and the change in pressure, these being unknown, any quotient obtained by the division of the amount of fluid removed by the difference in pressure, or the reciprocal of this quotient, such as Ayala's rachidial quotient, will be only an arbitrary figure, perhaps of some clinical use but in no way indicative of the amount of fluid present in the cerebrospinal fluid system.

27. Henderson, Yandell, and Haggard, W. H.: Circulation in Man in the Head Down Position and a Method for Measuring the Venous Return to the Heart, J. Pharmacol. & Exper. Therap. **11**:189, 1918.

28. Clark, Janet H.; Hooker, Donald, and Weed, L.: The Hydrostatic Factor in Venous Pressure Measurements, Am. J. Physiol. **109**:166, 1934.

The bulk modulus, $\frac{\frac{dP}{dV}}{V}$, cannot be used in determining the elasticity of the meningovascular containers of the cerebrospinal fluid. Since their areas and thickness are unknown, the quotients obtained under different conditions vary and are only empirical figures.

When accurate measurement of the cerebrospinal fluid pressure is desired open manometers should not be used. Certain reasons for their inaccuracy are discussed.

When through combined cistern and lumbar punctures pressures were read in two open manometers and a tilt was made, little or no fluid was found to enter the dural space, provided that the zero points of the two manometers were placed opposite their respective entrances into the subdural space. The only factor which we found could be studied by a double manometric reading was the pressure necessary to push back into the subdural space a certain amount of fluid in a manometer by changing the relationships of another manometer.

Certain fallacies arising from the concept that the cerebral contents are enclosed in a rigid or closed box are discussed. These relate to changes in pressure on tilting, the production of negative pressure, contractility of the blood vessels, strain on blood vessels and cerebral circulation.

Changes in cerebrospinal fluid pressure by tilting are due to the resultant of forces, among them vascular reflection and hydrostatic pressure, and not to the greater force.

The cerebrospinal contents are exposed to atmospheric pressure only through the blood vessels. The difference in the changes of cerebrospinal fluid pressure in the head-down and tail-down positions is due to the difference in pressures in the blood vessels in these respective positions.

RADICULONEURITIS WITH ACELLULAR HYPERALBUMINOSIS OF THE CEREBROSPINAL FLUID

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My purpose in this article is to discuss the neurologic syndrome which, in collaboration with J. A. Barré and A. Strohl, was described [1] for the first time in 1916. Since, numerous cases have been reported in different countries under the title syndrome of Guillain-Barré or some other name. I propose to give a résumé of ten unpublished cases. Furthermore, I shall delimit as exactly as possible the syndrome which we isolated so that it may be kept distinct from the broad group of polyradicular neuritides which occur commonly in neurologic practice.

In the original report [1] to the Société médicale des hôpitaux de Paris on Oct. 13, 1916, we said:

> We call attention to a clinical syndrome which we have observed in two cases, a syndrome characterized by motor disturbances, loss of tendon jerks, with preservation of cutaneous reflexes, paresthesias with slight disturbance of objective sensibility, tenderness on pressure of the muscles, little change in the electrical reactions of the nerves or muscles and noteworthy hyperalbuminosis of the cerebrospinal fluid in the absence of cytologic reaction (albuminocytologic dissociation). This syndrome seems to us to result from a concomitant attack on the spinal roots, nerves and muscles, probably by an infectious or toxic agent. It must be differentiated from simple radiculitis, pure polyneuritis and polymyositis. Graphic studies of the speed of the reflexes and their latent time and of the character of the muscular contractions demonstrate that the entire neuromuscular apparatus participates in the syndrome. We stress particularly the acellular hyperalbuminosis of the cerebrospinal fluid, an observation which, as far as we know, has not previously been described in similar cases.

We added:

> The prognosis does not appear to be extremely serious, if we may judge from the course of the disease in our two patients; the first had almost recovered and the second was improving when they were discharged from the army.

Permit me to emphasize that the observation of pronounced hyperalbuminosis of the cerebrospinal fluid in the absence of cellular reac-

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1. Guillain, Georges; Barré, J. A., and Strohl, A.: Sur un syndrome de radiculo-névrite avec hyperalbuminose du liquide céphalo-rachidien sans réaction cellulaire: Remarque sur les caractères cliniques et graphiques des réflexes tendineux, Bull. et mém. Soc. méd. d. hôp. de Paris **40**:1462 (Oct. 13) 1916.

tion in the syndrome of radiculoneuritis was new in 1916; at that time, twenty years ago, albuminocytologic dissociation had been described only in association with compression of the spinal cord and with Pott's disease.

Our first communication established in cases of polyradiculoneuritis of this type, first, the albuminocytologic dissociation in the fluid, and, second, the favorable prognosis. I have insisted repeatedly on these two characteristics of the syndrome: I did so [2] in March 1925, in a communication to the Réunion neurologique de Strasbourg, in collaboration with Alajouanine and Périsson. Furthermore, on the basis of these criteria a number of neurologists have reported similar observations under the title *syndrome Guillain-Barré*.

The most important of the later reports of observations of the syndrome are those of Marie and Chatelin,[3] Govaerts,[4] Fornara,[5] Draganescu and Claudian,[6] Metzger,[7] Krakowski and Poncz,[8] Boeff,[9] François, Zuccoli and Montus,[10] Hendrickx,[11] Marinesco and Draganescu,[12] Kuhl-

2. Guillain, Georges; Alajouanine, T., and Périsson, J.: Sur le syndrome de radiculo-névrite aiguë curable avec dissociation albuminocytologique du liquide céphalo-rachidien, Rev. neurol. **1**:492, 1925.

3. Marie, Pierre, and Chatelin, C.: Note sur un syndrome de paralysie flasque plus ou moins généralisée avec abolition des réflexes, hyperalbuminose massive et xanthochromie du liquide céphalo-rachidien, évoluant spontanément vers la guérison et de nature indéterminée, Rev. neurol. **2**:564, 1916.

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5. Fornara, Piero: Contributo allo studio delle meningo-radicoliti, Clin. pediat. **9**:403, 1927.

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9. Boeff: Un cas de polyradiculonévrite curable: Syndrome de Guillain et de Barré, Medicinsko spisanie, 1928, vol. 12, no. 10.

10. François; Zuccoli, G., and Montus, G.: Sur un cas de polyradiculonévrite curable avec dissociation albumino-cytologique: Syndrome de Guillain et de Barré, Rev. neurol. **1**:95, 1929.

11. Hendrickx, H.: Polyradiculo-névrite avec dissociation albumino-cytologique et paralysie faciale double, J. de neurol. et de psychiat. **29**:584, 1929.

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mann,[13] Chavany and Thiébaut,[14] Patrikios,[15] Draganescu and Façon,[16] Alfandary and Stayitch,[17] von Sántha,[18] Mathon,[19] Camaüer and Schultz Ortiz,[20] Riser and Planques,[21] Riser and Sol,[22] Babonneix and Lévy,[23] Metzger and Mandel,[24] Meyer,[25] Baruk and Poumeau-Delille,[26] Popek,[27] Abel, Kissel and Simonin [28] and Boudin.[29]

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13. Kuhlmann, M.: Syndrome de Guillain-Barré, Encéphale **26**:563 (July-Aug.) 1931.

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19. Mathon, K.: Deux cas de polyradiculonévrite avec dissociation albumino-cytologique: Syndrome de Guillain-Barré, Rev. neurol. **2**:165, 1934.

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22. Riser, M., and Sol: De la névraxite zostérienne: Lésions du système nerveux central dans la zona, Encéphale **28**:380, 1933.

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(Footnote continued on next page)

REPORT OF CASES

The first five reports are concerned with cases of polyradiculoneuritis with albuminocytologic dissociation involving all the limbs, but not the cranial nerves.

CASE 1.—L., a man aged 58, who was admitted to the neurologic clinic of the Hospital of the Salpêtrière in October 1923, became ill in May 1923 with paresthesias of the hands and feet. This was followed by progressive paresis of all extremities.

Examination.—The findings included: 1. Paralysis, which was especially pronounced in the flexor muscles of the leg and the extensor muscles of the toes and fingers. 2. Loss of all tendon and periosteal reflexes in all limbs; preservation of cutaneous reflexes, and exaggeration of idiomuscular contractions on percussion. 3. Extreme tenderness to pressure of the muscles and stretching of the nerves (Lasègue's sign); no change in superficial sensibility, and severe disturbance of deep sensibility (sense of position and vibration). 4. Spinal fluid: clear fluid; albumin, 0.85 Gm. per hundred cubic centimeters; cells, 0.9 per cubic millimeter, and Wassermann reaction, negative.

Course.—With therapy with the antiseptic remedies sodium salicylate and methenamine, improvement was progressive; complete recovery ensued in a few weeks.

CASE 2.—Mme. P., aged 45, who was admitted to the neurologic clinic of the Salpêtrière on Nov. 23, 1931, had experienced lumbar pain and electrical sensations in the feet at the end of September 1931. In the early part of October paralytic phenomena appeared in the lower extremities, with resultant difficulty in walking and falling because the legs gave way. Examination at a hospital in Paris revealed: flaccid paralysis of the legs, slight diminution in the electrical reactions of the muscles and absence of changes in objective sensibility. The spinal fluid contained 0.56 Gm. of albumin per hundred cubic centimeters and 1.5 cells per cubic millimeter; the Wassermann reaction was negative.

Examination.—The findings included: 1. Paresis of all muscles of the legs, which was especially pronounced in the flexors of the knee; general loss of muscular power in the arms, and hypotonia without muscular atrophy. 2. Slight diminution in the electrical excitability of the muscles and nerves in all limbs. 3. Loss of tendon and periosteal reflexes in all limbs; absence of the plantar reflexes, and exaggeration of idiomuscular contractility on percussion, which in some muscles resulted in actual contracture. 4. Extreme tenderness on compression of the muscles; no alteration in superficial or deep sensibility. 5. Spinal fluid: clear fluid; pressure, 56 cm. of water, with the Claude manometer, when the patient was in a sitting posture; albumin, 0.4 Gm.; Pandy reaction, positive; Weichbrodt and Wassermann reaction, negative; 1.3 lymphocytes per cubic millimeter, and colloidal benzoin reaction, 0000022221000000.

Course.—Injections of sodium salicylate and methenamine were given; electrical therapy with ionization of potassium iodide was also employed. Improvement was rapid. When discharged from the hospital in February 1932, the patient had completely recovered, though the tendon jerks were still absent.

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29. Boudin, G.: Les polyradiculonévrites généralisées avec dissociation albumino-cytologique: Etude anatomo-clinique et considérations sur les infections à virus neurotrope touchant avec prédilection les nerfs, Paris, Norbert Maloine, 1936.

CASE 3.—C., a man aged 39, who was examined by me in December 1931, had for some months previously experienced difficulty in walking and later motor disturbances in the upper limbs.

Examination.—The findings included: 1. Complete loss of power in the lower extremities, with the maximum loss in the small muscles of the foot and the posterior and antero-internal portions of the leg (internal and external popliteal nerves). Muscular power was generally deficient in the upper extremities, the defect being more pronounced in the muscles of the forearm than in those of the upper portion of the arm and greatest in the thenar, hypothenar and interosseous muscles of the hands. The thenar and hypothenar muscles were slightly atrophied. Fibrillary twitchings were present in the thenar eminences and also in muscles of the lower extremities, particularly the quadriceps muscles. 2. Loss of all tendon and periosteal reflexes in all limbs; normal cutaneous reflexes. 3. Partial reaction of degeneration in the muscles innervated by the external and internal popliteal nerves on both sides; slight reaction of degeneration in the muscles of the antero-external and posterior portions of the lower parts of the legs, the plantar muscles being the most affected. In the upper limbs there was a slight reaction of degeneration in the muscles of the hands. After electrical stimulation the fibrillary twitchings became more evident. 4. Paresthesias in the limbs; cramps in the arms and legs; tactile, painful and thermic hypesthesia of the feet and hands, and severe disturbance of deep sensibility, with astereognosis in the arms and loss of sense of vibration and of position in all limbs. The change in deep sensibility probably explained a certain degree of ataxia which was noted in the upper extremities. 5. No disturbance of sphincteric function. 6. Spinal fluid: clear fluid; albumin, 0.5 Gm.; Pandy reaction, positive; 4 cells per cubic millimeter; Wassermann reaction, negative, and colloidal benzoin reaction, 0000002221000000. 7. Negative Wassermann reaction of the blood.

Prognosis.—Although physicians who had examined the patient had given an unfavorable prognosis, I predicted a favorable outcome.

Course.—Treatment with sodium salicylate and methenamine was prescribed; to this was added administration of strychnine and vitamins. Transcerebromedullary applications of electrolyzed iodide were combined with the application of infra-red rays, white light and diathermy to the extremities. Improvement was slow but progressive; recovery of both motor and sensory functions was complete. It should be noted that in March 1933, almost two years after the onset of the disease, loss of tendon reflexes still persisted as the only sequel of the illness.

CASE 4.—Roger G., a man aged 22, who was admitted to the neurologic clinic of the Salpêtrière on Feb. 22, 1934, had experienced severe pain in the lumbar region while skating on Jan. 21, 1934; he rested for a few minutes and resumed skating. A few days later there appeared difficulty in walking, pain in the muscles of the buttocks and of the lumbar region and difficulty in contracting the gluteus muscles. He complained of formication in the hands and of an electrical sensation in the lumbar region during effort. About the first of February there appeared genito-urinary disturbances, loss of power of erection and inability to stop the flow of urine during micturition.

There had been no previous illness of importance. The present illness had arisen without apparent cause and without evidence of infection, sore throat or intestinal disturbance.

Examination.—The findings included: 1. Difficulty in walking and diminished muscular power, involving particularly the flexors of the knee and hip and the

gluteus muscles. The distal muscles of the limbs did not seem to be affected. There was no motor involvement of the upper extremities. 2. Loss of the patellar, achilles, medioplantar, posterior tibiofemoral and posterior peroneofemoral reflexes; weakness of the styloradial and triceps reflexes in the upper extremities; normal plantar reflexes; loss of the cremasteric reflexes, and preservation of abdominal reflexes, except for the reflex in the left inferior quadrant. 3. Paresthesias of the hands; tenderness on compression of the muscles, and no objective disturbance of superficial or deep sensibility. 4. Loss of the power of erection of the penis; slight disturbance in function of the vesical sphincter. 5. Spinal fluid: clear fluid; pressure, 10 cm. of water with the patient in the recumbent position; albumin, 1.07 Gm.; Pandy reaction, positive; Weichbrodt reaction, positive; 7 lymphocytes per cubic millimeter; Wassermann reaction, negative; colloidal benzoin curve, 0000012122210000. 6. Negative Wassermann reaction of the blood.

Course.—Therapy consisted of intravenous injections of sodium salicylate and sodium iodide, intramuscular injections of methenamine and administration of colloidal silver by mouth. Transcerebromedullary ionization with iodide was also given.

Lumbar puncture in May 1934 revealed: clear fluid, under a pressure of 22 cm. of water; albumin, 1.7 Gm.; Pandy reaction, positive; Weichbrodt reaction, negative; 1.3 lymphocytes per cubic millimeter, and colloidal benzoin reaction, 0012220221000000.

Clinical improvement was rapid from the time when treatment was initiated. In June the patient had practically recovered; he walked normally and could ride 10 kilometers on a bicycle; muscular power was normal; all reflexes were normal, and the genito-urinary disturbances had disappeared.

Case 5.—Mme. Mathilde P., aged 35, was admitted to the neurologic clinic of the Salpêtrière on April 10, 1934, two months after parturition complicated by bilateral phlebitis, from which she had recovered. Toward the middle of February 1934 she had experienced sensations of numbness in the lower extremities and the spine, with severe pain in all limbs. About the middle of March numbness appeared in the upper extremities, and the hands became paralyzed. Paresthesias were present in the face, mouth and tongue.

Examination.—The findings included: 1. Complete flaccid paralysis of the lower extremities, with hypotonia; diminution of muscular power in the upper limbs, and slight atrophy of the thenar muscles. 2. Loss of tendon and periosteal reflexes in all limbs; preservation of cutaneous reflexes, and loss of corneal reflexes. 3. Tenderness of muscles to pressure; slight diminution of tactile, painful and thermal sensibility in the lower extremities, and severe disturbances of deep sensibility, including sense of vibration and of position, in all limbs; complete astereognosis in the hands, and dysmetria and adiadokokinesis in the upper limbs, due undoubtedly to the disturbance of deep sensibility. 4. Difficulty of micturition. 5. Spinal fluid: clear fluid; pressure, 16 cm. of water, with the patient in the recumbent position; Weichbrodt and Wassermann reactions, negative; Pandy reaction, positive; 0.5 lymphocytes per cubic millimeter; colloidal benzoin reaction 0000012000222000. 6. Negative Wassermann reaction of the blood.

Course.—Antiseptic remedies and transcerebromedullary ionization with iodide were employed. Improvement was progressive, and clinical recovery was complete in six months.

The patient was reexamined at the Salpêtrière in June 1936, two years after recovery. A physician who had noted the loss of tendon jerks in the lower

limbs had regarded the case as one of tabes and had prescribed mercuric cyanide and sulfarsphenamine, administration of which had not been well borne. My examination revealed that, though the knee jerks were normal, the achilles and medioplantar reflexes were absent. Apart from this, there was no evidence of the illness of 1934.

The following cases illustrate involvement of the cranial nerves in addition to that of the roots and nerves of the extremities. Recovery, however, was as complete as that in the preceding cases.

CASE 6.—Mme. G., aged 48, was admitted to the neurologic clinic of the Salpêtrière on March 31, 1927, with difficulty in walking, speaking and swallowing. On March 28, without preceding infectious illness, her voice had suddenly become nasal, and fluids had regurgitated through the nose. On the same day ptosis developed on the left. On the next day she experienced formication in the hands and feet, and in the afternoon she became incapable of walking unassisted because of weakness of the legs. On March 30 she had an involuntary stool and complained of numbness in the genito-urinary region.

Examination.—The findings included: 1. Ataxic gait, the patient not being able to feel the ground and having the sensation of walking on rubber, and global diminution of muscular power in the lower extremities, affecting especially the proximal muscles. 2. Loss of tendon reflexes in all the limbs, and preservation of all cutaneous reflexes. 3. Paresthesia of the distal parts of the limbs and of the perineal region; no objective alteration of superficial or deep sensibility. 4. Disturbances involving the cranial nerves: bilateral ptosis, which was more marked on the left; paresis of the palate, and defective sense of taste. There was no alteration of the pulse or respiration. 5. Slight elevation of temperature, which continued for several days. 6. Spinal fluid: clear fluid; pressure, 32 cm. of water, with the patient in the sitting position; albumin, 0.4 Gm.; Pandy reaction, positive; Weichbrodt and Wassermann reactions, negative; 0.5 cells per cubic millimeter; colloidal benzoin reaction curve, 0010222222000000. 7. Sterile cultures of material from the nasopharynx for diphtheria bacilli.

Course.—Injections of sodium salicylate and methenamine were given. Recovery was complete in a few weeks.

CASE 7.—Mme. L., aged 50, was admitted to the neurologic clinic of the Salpêtrière on April 11, 1931, with difficulty in walking and paresthesias of the hands. The illness began in February 1931 with pain in the right leg, resembling sciatic neuralgia. By the end of March there were weakness of the lower extremities, uncertainty in walking, pain in the upper extremities and paresthesias of the hands.

Examination.—The findings included: 1. Ataxic gait and slight diminution of power in the muscles of all the limbs. 2. Bilateral facial paresis. 3. Loss of all periosteal and tendon and corneal reflexes. 4. Partial reaction of degeneration in all the facial muscles and in all muscles of the upper extremities, being most pronounced in the hands; slight reaction of degeneration in the distribution of the external popliteal nerve on both sides. 5. Tenderness to pressure of the muscles of the lower extremities; no objective alteration of superficial or deep sensibility. 6. Spinal fluid: clear fluid; pressure, 25 cm. of water, with the patient in the recumbent position; albumin, 1.07 Gm.; Pandy reaction, strongly positive; Weichbrodt reaction, positive; 3 lymphocytes per cubic millimeter; Wassermann reaction, negative; colloidal benzoin reaction, 0000002222210000. 7. Negative Wassermann reaction of the blood.

Course.—Injections of sodium salicylate and methenamine and electrical applications were given. Recovery was complete in a few months.

CASE 8.—Camille D., a man aged 49, who was admitted to the neurologic clinic of the Salpêtrière on May 9, 1934, had experienced paresthesias of the feet and hands and weakness of the right leg on March 13, 1934. Paralysis of the left side of the face appeared on March 19. On March 20 and 21 there were painful cramps of the muscles of all limbs and of the lumbar region. A few days later the extremities were all paralyzed, and at this time cardiac difficulties occurred, with palpitation and dyspnea. When the patient was admitted to the hospital the condition had already improved.

Examination.—The findings included: 1. Paresis of the lower limbs, the weakness being as great in the flexor as in the extensor muscles, and slight paresis of the left side of the face. 2. Loss of tendon and periosteal reflexes in all extremities and preservation of all cutaneous reflexes. 3. No alteration in superficial or deep sensibility. 4. Spinal fluid: clear fluid; pressure, 40 cm. of water, with the patient in the sitting position; albumin, 0.40 Gm.; Pandy reaction, slightly positive; Weichbrodt and Wassermann reactions, negative; 0.8 lymphocytes per cubic millimeter; colloidal benzoin reaction, 0000012100000000. 5. Negative Wassermann reaction of the blood.

Course.—Injections of sodium salicylate and methenamine and electrical treatments were given. Recovery was complete one month later, though the tendon reflexes remained absent in all the extremities.

CASE 9.—M., a man aged 21, was hospitalized in a clinic in Paris in November 1934 because of paralytic disturbances in all limbs. The illness began in July 1934, when he noticed difficulty in running while playing tennis. Paresis of the legs increased in August, movements of the feet and toes being particularly difficult; the hands became clumsy; paresthesias as well as hyperesthesia were experienced in the feet. A physician noted loss of tendon jerks and performed a lumbar puncture. The fluid was clear; the pressure was 12 cm. of water, with the patient in the sitting position; the albumin content was 0.71 Gm.; the Nonne reaction was positive; the chloride, dextrose and urea contents were 6.91, 0.71 and 0.26 Gm., respectively, per hundred cubic centimeters of fluid; the cell count was 1 lymphocyte per cubic millimeter; the Wassermann reaction was negative, and the colloidal benzoin reaction was 0000022002222100. Paralysis increased in both the upper and the lower extremities, and the patient was placed in a hospital.

Examination.—The findings included: 1. Almost complete paralysis of the legs, the muscles of the thighs and the gluteus muscles still contracting feebly. In the upper extremities the motor disturbance affected most the thenar, hypothenar and interosseous muscles; the muscles of the anterior aspect of the forearm were more affected than those of the posterior. 2. Reaction of all muscles of the lower limbs to the faradic current, both through the nerve and directly. There was, however, diminished amplitude of contraction, especially in the muscles of the legs and feet, and contractions to galvanic stimulation were all slow, though only slightly so. Thus, there was definite diffuse partial reaction of degeneration. All muscles of the upper limbs reacted to faradism, both through the nerve and directly, but with diminished amplitude of contraction; there was also distinct slowness of contraction on galvanic stimulation of the biceps, triceps and supinator brevis muscles and particularly the muscles of the hand. The diffuse partial reaction of degeneration was less distinct in the lower than in the upper extremities. 3. Loss of tendon and periosteal reflexes in all limbs; preservation

of cutaneous reflexes. 4. Spontaneous pain and severe tenderness on pressure of the muscles; tactile, painful and thermic hypesthesia in the extremities, which was more pronounced in the feet than in the hands; distinct alteration of deep sensibility, including sense of position and of vibration, especially in the lower extremities, and astereognosis of the hands.

Course.—Methenamine and colloidal gold and silver were administered, and transcerebromedullary ionization with iodine and diathermy were used. Slight but progressive improvement occurred until December 1934, when there was an exacerbation. About December 12 a febrile state developed and continued until Jan. 15, 1935. Cultures of the blood remained sterile. Phlebitis of the left femoral vein, with local rise of temperature and edema, became evident at the beginning of January. At about the same time there was aggravation of the neurologic symptoms.

Examination at this time revealed: 1. Severe pain in all limbs and in the trunk, rendering touch, and especially pressure, extremely painful. 2. Almost complete anesthesia of the feet, legs and hands. The alterations in deep sensibility already noted were accentuated. 3. Complete disappearance of the recently acquired spontaneous movements of the toes; the muscles of the thighs could hardly be contracted. Strength in the muscles of the hands, in the extensor muscles of the fingers and of the wrists diminished so much that the hands dropped as in bilateral paralysis of the radial nerve and became completely helpless. 4. Complaint of diplopia. Lagrange reported the presence of affection of the right oculomotor nerve, with paresis of the internal and superior rectus muscles on the right. 5. Difficulty in breathing at times.

Electrical examination in February, when the paralysis was at the maximum, revealed much more favorable responses than the clinical examination would have led one to expect. All muscles, even the most paralyzed, still reacted to faradism, both through the nerves and directly; everywhere the response to galvanic stimulation was slow, though a few fibers still reacted quickly. These electrical tests tended to prove that the loss of function was due to changes in conductivity and not to destruction of nerve fibers and permitted one to affirm the favorable clinical prognosis which had previously been given, in spite of the extent and severity of the paralysis.

Use of antiseptic remedies (methenamine, a colloid silver solution and quinine) were continued throughout the febrile period. To this were added the administration of strychnine, extracts of adrenal cortex and mesothorium and electrotherapy in the form of ionization of iodide, given at first spinoperipherally and later transcerebrospinally.

Improvement in the clinical symptoms was slow but progressive in the succeeding months. In December 1935 recovery was complete; there remained no sequelae, even the reflexes having returned to normal.

Case 10.—D., a man aged 50, who was admitted to the neurologic clinic of the Salpêtrière on March 23, 1935, had become ill in February 1935, with formication and numbness in the hands. Several days later he had experienced difficulty in walking.

Examination.—The findings included: 1. Inability to walk; severe and global loss of power in the muscles of all extremities, and slight atrophy of the muscles of the legs and forearms. 2. Relatively slight changes in the electrical excitability of muscles and nerves of the limbs. 3. Loss of tendon reflexes in all limbs, and preservation of cutaneous reflexes. 4. Paresthesias of the extremities; tenderness on pressure of the muscles; little change in tactile, painful or thermic sensibility, with possibly slight cutaneous hyperesthesia; severe disturbance of deep sensibility,

including sense of position and of vibration, and definite ataxia of all limbs, which was due probably to the loss of deep sensibility. 5. Cranial nerves intact. 6. Spinal fluid: clear fluid; pressure, 24 cm. of water; albumin, 0.71 Gm.; Pandy reaction, positive; Weichbrodt and Wassermann reactions, negative; no cells, and colloidal benzoin reaction curve, 0000022222100000. 7. Negative Wassermann reaction of the blood.

Course.—In the beginning of April bulbar signs appeared: paresis of the left side of the face, difficulty in swallowing, tachycardia and sweating. At the same time the paralytic phenomena became accentuated. The bulbar symptoms, which at times seemed serious, lasted for several weeks and then disappeared.

During the patient's stay in the hospital, sodium salicylate and methenamine were administered. Improvement was already evident in May. At the time of discharge, on July 23, 1935, power had returned in all extremities, though the tendon reflexes of the legs were still absent.

COMMENT AND SUMMARY

I shall now summarize briefly the clinical symptomatology, evolution, diagnosis and treatment of this syndrome.

Symptomatology.—The disease occurs in both sexes and most often in persons between the ages of 20 and 50; in children and old persons it is much more rare.

I am convinced that the disease is of infectious origin, though, as in many infections with a neurotropic virus, fever is often absent, or at least is slight. In some cases there have been sore throat, digestive disturbance, stiffness of muscles or general malaise before the onset of the motor or sensory symptoms. Although in some cases it has been noted that the polyradiculoneuritic syndrome developed after a suppurative infection, I am of the opinion that in such instances a neurotropic virus plays the principal rôle but acquires its virulence as the result of the primary illness.

Sometimes the onset is abrupt, being manifested from the start by attacks of pain, paresthesia or paralytic phenomena.

During the height of the disease the symptoms are those described in our first report,[1] in 1916.

The motor disturbances are progressive and do not have the abrupt, global character of those associated with acute anterior poliomyelitis, the Heine-Medin disease. Most often they involve first the muscles of the lower extremities and later those of the trunk and upper extremities, giving rise sometimes to complete quadriplegia. However, the paralysis or paresis is rarely equally severe in the whole extent of the limbs; it affects electively the muscles of the ends of the limbs (lower portions of the legs and feet and the forearms and hands), as in polyneuritis. In the upper limbs the paralysis is often circumscribed. In rare cases the paralysis predominates in the proximal muscles of the limbs. The phrenic nerve is rarely affected.

The paralysis is flaccid and is usually associated with hypotonia. However, in one of the first cases observed in 1916 my colleagues and I were struck by the fact that some degree of hypertonia existed in addition to the paralysis, and we commented that this state of hypertonia was due not to meningitis but to some alteration in contractility which seemed to depend on damage of the peripheral nerve; we stated also that conditions of hypertonia may be observed in the course of certain forms of peripheral neuritis and in cases of incomplete wound of a nerve. In some instances I have observed fibrillary twitchings.

Muscular atrophy is much less marked than in the majority of forms of infectious or toxic polyneuritis which have been previously recognized.

In several instances (case 1 reported in our first paper [1] in 1916 and cases 3, 5, 6 and 10 of the present series), I observed the presence of ataxia, which has been noted also by other authors. Some patients may present a pseudotabetic appearance like that associated with diphtheritic neuritis.

Alteration of electrical reactions, including both the classic reactions to faradic and galvanic stimulation and the chronaxic reactions, is slight in this syndrome. This fact is worth bearing in mind.

The tendon and periosteal reflexes are abolished in the domain of the paralyzed muscles, and this abolition may extend to regions which are apparently intact. It will be noted that in the greater number of the cases reported there was loss of the patellar, achilles, medioplantar, posterior tibiofemoral and adductor reflexes in the lower extremities and of the styloradial, cubitopronator, radiopronator, triceps, flexor, acromial and scalpular reflexes in the upper extremities. In some cases I have observed preservation of midline reflexes (mediopubic and mediosternal) concomitantly with loss of the tendon and periosteal reflexes of the limbs. Abolition of the tendon and periosteal reflexes is an important sign and may persist for a long time after recovery from the paralysis.

Idiomuscular contractility to percussion is conserved, as was noted in the first two patients observed in 1916. Sometimes, even, it may be exaggerated.

Cutaneous reflexes (plantar, cremasteric and abdominal), in contrast to the tendon jerks, are usually normal, though finally they may also be abolished.

The sensory changes are: spontaneous pain, sometimes of extremely violent character at the start, and pain provoked by pressure on the muscles and nerves or by stretching the nerves (sciatic and crural nerves and the brachial plexus). In several cases I observed painful cramps, especially in muscles of the lower extremities. There are also par-

esthesias, formication, numbness and sensations of burning and of electrical stimulation in the feet and hands.

Objective sensory changes are frequently slight, such as tactile, painful and thermic hypesthesia or hyperesthesia. These are most pronounced at the periphery of the limbs. Changes in deep sensibility (sense of position and of vibration) are not uncommon. In several cases I observed astereognosis. Changes in deep sensibility may explain the ataxia that is sometimes present. Most of these disturbances are transitory and disappear rapidly.

In case 2 reported in 1916, the patient was able to urinate without aid but did not perceive the passage of the urine; in several instances since, I have observed difficulty and slowness in micturition and loss of perception of the passage of the urine. However, disturbance of function of the vesical sphincter is unusual, and the lower sacral roots are rarely involved.

Sexual impotence in the male, in the instance (case 4) in which it was observed, was transitory.

To the clinical signs of damage of spinal roots may be added paralysis of cranial nerves. The facial nerve appears to be especially susceptible; paralysis of this nerve was present in cases 7, 8 and 10 in this series and in other cases reported in the literature by Govaerts,[4] Fornara,[5] Boeff,[9] François, Zuccoli and Montus,[10] Hendrickx,[11] Mathon,[19] Metzger and Mandel[24] and Boudin.[29] The paralysis is of peripheral type and usually slight and transitory and disappears completely. In a case reported by Alajouanine and Boudin, the facial palsy left as a sequel facial hemispasm.

It should be realized that there are cases of polyneuritis associated with facial diplegia which do not belong to the syndrome under discussion and in which the etiology is different. Instances reported by Viets[30] and by Taylor and McDonald[31] appear to belong with the cases described in this paper.

Diplopia due to paralysis of ocular muscles is relatively rare; it disappears rapidly; I have observed it in two cases.

Involvement of the trigeminal, glossopharyngeal, vagus, spinal accessory and hypoglossal nerves is uncommon. The major bulbar disturbances, with changes in phonation, swallowing and respiratory and cardiac rhythm, are exceptional; they are transitory and disappear completely.

Examination of the cerebrospinal fluid gave the principal characteristics which enabled us to distinguish this syndrome. The fluid was

30. Viets, Henry R.: Acute Polyneuritis with Facial Diplegia, Arch. Neurol. & Psychiat. **17**:794 (June) 1927.

31. Taylor, E. W., and McDonald, G. A.: The Syndrome of Polyneuritis with Facial Diplegia, Arch. Neurol. & Psychiat. **27**:79 (Jan.) 1932.

clear in all the cases in my series. Xanthochromia, which has been reported, is infrequent. Hyperalbuminosis is constant and pronounced; the albuminoids usually amount to from 1 to 2 Gm. per hundred cubic centimeters, sometimes more. Cases with slight hyperalbuminosis, with an albuminoid content of from 0.3 to 0.4 Gm., do not belong to the syndrome or must be regarded as instances of an abortive form. The Pandy reaction is more commonly positive than the Weichbrodt reaction. In contrast to the hyperalbuminosis, there is no hypercytosis; in other words, there is albuminocytologic dissociation. I refuse to recognize radiculoneuritis with hyperlymphocytosis or hypernucleosis as belonging to this syndrome. The Wassermann reaction is always negative.

The colloidal benzoin reaction is frequently changed, as I reported in 1925,[2] in collaboration with Alajouanine and Périsson. In cases of polyradiculoneuritis one observes frequently an increase of precipitation in the meningitic zone. This precipitation has no relation to the amount of hyperalbuminosis as a whole, as has wrongly been suggested, but is due to the presence of certain globulins possessing a particular electrical charge, which is derived from disintegration of nerve tissue.

The hyperalbuminosis diminishes rapidly in a few weeks, though one may observe slight hyperalbuminosis for a long time.

Prognosis.—The outlook for recovery is favorable. All patients I have observed recovered, and practically all authors have reported the same result. Two cases in which the outcome was fatal, reported by Margulis and by Alajouanine, Thurel, Hornet and Boudin, do not belong to this group. The acute bulbar forms of Landry's paralysis, likewise, are dissimilar. The recognition of the benignity of the prognosis is of real importance.

Incidentally, mention may be made of case 9, in which phlebitis complicated by two pulmonary emboli developed in the evolution of the disease. Phlebitis occurred also in a case observed by Kuhlmann and in one described by Boudin. It is impossible to say exactly whether these attacks of phlebitis were caused by the same infection as that producing the polyradiculoneuritis or by some additional infection.

The disease may last for weeks or months; recovery is usually complete, without paralytic sequelae or muscular atrophy. Paresthesias may persist for a long time, with slight fatigability on walking. The reflexes often remain absent for a long time (cases 2, 3, 5 and 8), without causing the patient the least difficulty. Recently, I reported to the Société de neurologie de Paris [32] the case of a young woman who

32. Guillain, Georges: Sur un cas de radiculo-névrite avec hyperalbuminose du liquide céphalo-rachidien sans réaction cellulaire: Guérison complète, mais persistance de l'abolition des réflexes tendineux; ses conséquences pour les diagnostics d'avenir, Rev. neurol. **1**:799, 1936.

had an attack of polyradiculoneuritis with albuminocytologic dissociation in 1930 and who recovered completely in a few months; she then passed through two normal pregnancies; five years after the original illness the tendon reflexes were still absent in all the limbs. It is important to recognize this fact, for if such patients, ten or fifteen years after the polyradiculoneuritis, suffer rheumatic or abdominal pain or some other symptom, physicians observing the absence of tendon reflexes and not being aware of the previous illness, reach inevitably the incorrect diagnosis of tabes, with all the consequences which this carries.

It is of value in every case to control the clinical observation of loss of tendon reflexes by use of the graphic method; in this way, in collaboration with Barré and Strohl,[1] I demonstrated that reflexes which were inelicitable with the clinical percussion hammer may still be detected in the graphic records. Such records afford exact information on the modalities of muscular contractions and the latent time of reflexes, through study of the myographic curves.

Diagnosis.—The recognition of the disease is usually easy. Differentiation must be made from the following diseases:

Acute anterior poliomyelitis, or the Heine-Medin disease. The onset in this disease is more acute; massive paralysis develops rapidly, and fever at the time of onset is higher. Objective sensory disturbances are lacking. In many cases during the first phase of poliomyelitis there is a pronounced meningeal reaction; signs of meningitis are lacking in the syndrome of polyradiculoneuritis. The spinal fluid in acute poliomyelitis presents nothing resembling the albuminocytologic dissociation characteristic of polyradiculoneuritis. The course of evolution of the atrophic palsy occurring in poliomyelitis is entirely different from that present in the syndrome under discussion.

Polyneuritis secondary to infection and intoxication: These are readily distinguished.

Acute febrile polyneuritis: These conditions, including the form reported by Gordon Holmes [33] during the World War, can be recognized, first, by the fever, which is lacking in the syndrome discussed in this paper, and, second, by the fact that the cerebrospinal fluid presents no abnormality, as was pointed out by Holmes. The same is true in the analogous cases reported by Bradford, Bashford and Wilson,[34] in which there were severe infection and considerable mortality.

Postdiphtheritic neuritis may be associated with albuminocytologic dissociation in the spinal fluid. In the report of a study in collaboration

33. Holmes, Gordon: Acute Febrile Polyneuritis, Brit. M. J. **2**:37 (July 14) 1917.

34. Bradford, J. B.; Bashford, E. F., and Wilson, J. A.: Acute Infective Polyneuritis, Quart. J. Med. **12**:88, 1918-1919.

with Laroche,[35] it was stated that in seven cases of diphtheritic paralysis observed at the Salpêtrière study of the cerebrospinal fluid revealed: hyperalbuminosis with the amount of albumin varying from 0.4 to 1.25 Gm., a positive Pandy reaction in six cases, though the Weichbrodt reaction was negative, and absence of hypercytosis. The evolution of diphtheritic paralysis, with the history of antecedent pseudomembranous sore throat, paralysis of the palate and disorders in accommodation, are entirely different. In the cases of polyradiculoneuritis with albuminocytologic dissociation in my series, I have frequently sought for diphtheria bacilli in the nasopharynx without success.

Syphilitic radiculitis has no relation to this syndrome; the diagnosis can readily be established by the findings in the cerebrospinal fluid—the positive Wassermann reaction and, frequently, hypercytosis. The cases of Bremer,[36] Delboeke and van Bogaert [37] and Trabaud [38] are instances of this condition.

Hence, polyradiculoneuritis associated with hyperalbuminosis and hyperlymphocytosis, without albuminocytologic dissociation, likewise do not belong to the syndrome I am discussing. Cases of the former condition have been reported by Riser, Labro and Planques;[39] Mussio-Fournier, Cervino, Rocca and Larrosa Helguera,[40] and Pommé, Tanguy and Marot.[41] Roger and Crémieux [42] observed radiculoneuritis with hypercytosis in association with Malta fever.

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35. Guillain, Georges, and Laroche, Guy: La réaction du benjoin colloïdal avec le liquide céphalo-rachidien de sujets atteints de paralysies diphtériques, Compt. rend. Soc. de biol. **113**:279 (May 20) 1933.

36. Bremer, F.: Polyradiculite avec coagulation massive spontanée et xanthochromie du liquide céphalo-rachidien, J. de neurol. et de psychiat. **20**:42, 1920.

37. Delboeke and van Bogaert, L.: Radiculo-névrite aiguë syphilitique avec syndrome de Froin, J. de neurol. et de psychiat. **25**:525 (Aug.) 1925.

38. Trabaud, J.: Syndrome de Guillain et Barré ou polyradiculo-névrite avec dissociation albumino-cytologique au cours d'une syphilis ignorée, Rev. neurol. **1**:808, 1929; Un nouveau cas de syndrome de Guillain et Barré d'origine spécifique, ibid. **2**:592, 1929.

39. Riser, Labro and Planques: De la méningo-neuronite primitive aiguë avec réaction méningée particuliérement intense (hypertrophie tronculaire, ataxie, papillite), Rev. neurol. **1**:1191, 1933.

40. Mussio-Fournier, J. C.; Cervino, José M.; Rocca, Francisco, and Larrosa Helguera, Rufino A.: Un cas de méningo-radiculo-névrite aiguë curable, avec xanthochromie et intense lymphcytose dans le liquide céphalo-rachidien, se terminant par une guérison complète, Rev. neurol. **2**:104, 1933.

41. Pommé, B.; Tanguy, R., and Marot, R.: Radiculo-névrite infectieuse à évolution régressive, Rev. neurol. **1**:749, 1934.

42. Roger, H., and Crémieux, Albert: La radiculo-névrite mélitococcique avec xanthochromie et réaction albumino-cytologique intense du liquid céphalo-rachidien, Marseille-méd. **66**:617 (Nov. 15) 1929.

I would include under this syndrome the case of Barker,[43] in which the cerebrospinal fluid contained an increased amount of albumin and globulin, while the cells never exceeded 10 per cubic millimeter and at certain examinations were absent. The condition described by Strauss and Rabiner [44] under the title myeloradiculitis may be allied to my syndrome of 1916, but these authors did not mention albuminocytologic dissociation of the spinal fluid.

Peripheral forms of the epidemic (lethargic) encephalitis of von Economo should not be confused with this syndrome; some authors have made this error in diagnosis. In cases of acute encephalitis I have never observed striking hyperalbuminosis of the spinal fluid without hypercytosis. The clinical manifestations of the so-called peripheral forms of epidemic (lethargic) encephalitis do not resemble in any way the rapidly curable syndrome of polyradiculoneuritis with albuminocytologic dissociation.

Treatment.—Polyradiculoneuritis with albuminocytologic dissociation is caused, I believe, by a neurotropic virus which undoubtedly will eventually be established through inoculation of susceptible animals; possibly animals in the surroundings serve as reservoirs of this virus, the manner of transmission of which, direct or indirect, is as yet unknown. Measures for prophylaxis, therefore, cannot be specified.

Antiseptic remedies should be prescribed; they seem to me to have a favorable influence. I recommend particularly intravenous injections of sodium salicylate (from 1 to 2 Gm. daily in serum containing 10 per cent of dextrose), intravenous or intramuscular injections of quinine, methenamine or colloidal silver and rubs with colloidal silver.

I believe that arsenicals are contraindicated; arsenic has unquestionably a harmful action.

It is well to combine with the antiseptic remedies electrotherapy in the form of ionization of iodine or calcium given by the transcerebromedullary path, warm baths and irradiation.

With these methods of therapy, because the virus of polyradiculoneuritis with albuminocytologic dissociation does not destroy nerve paths, progressive improvement and eventual recovery of the patient will be observed.

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43. Barker, Lewellys F.: Acute Diffuse (Cerebral and Spinal) Polyradiculoneuritis Following Oral Sepsis: Probability of Superimposed Infection with Neurotropic Ultravirus of Schwannophil Type, Arch. Neurol. & Psychiat. **31**:837 (April) 1934.

44. Strauss, Israel, and Rabiner, Abraham M.: Myeloradiculitis: A Clinical Syndrome with Report of Seven Cases, Arch. Neurol. & Psychiat. **23**:240 (Feb.) 1930.

AGGRESSIVE-SUBMISSIVE BEHAVIOR AND THE FRÖHLICH SYNDROME

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In attempting to correlate physical findings with personality traits, I have been impressed with the frequent occurrence of behavior problems characterized as submissive in children with the so-called Fröhlich syndrome. This impression was thought worthy of investigation, especially since in a behavior clinic for children problems in aggressive behavior are the rule.

The difficulty in correlating physical make-up and personality starts with the problem of clearly determining the physical signs and the personality traits to be measured. The difficulty is considered greater for the traits than for the physical signs. If observations on the latter include quantitative data and evidence of the presence or absence of any given item (for example, body weight and measurements, presence or absence of pubic hair, etc.) the differentiation is clearcut. When the physical signs are descriptive, their value as research data depends on a number of variable factors, including the examiner's experience, accuracy of observation and precision in the use of language, in addition to certain emotional factors that influence observation. In the Fröhlich syndrome, for example, one examiner may describe the obesity as typically of the mons-mammary-girdle type, the fingers as tapering and the skin as smooth and velvety, while another may describe the same patient in similar terms, qualified by the words "somewhat," "slight tendency to," "some evidence of" and the like, while yet another may check these items as normal, refusing to commit himself unless the signs are especially marked. In order to increase the accuracy of selection, only the cases were accepted in which the examiner was definite in his description of physical signs. Of the patients at the Institute for Child Guidance, 33 boys with the Fröhlich syndrome were found to satisfy these requirements.

It will be seen from a survey of the appended records that the diagnosis of the Fröhlich syndrome was based most frequently on the distribution of fat. At least, the three examiners at the Institute did not make a diagnosis of this condition unless that sign was present, regardless of other findings. This report might, therefore, be more accurately entitled "a study of certain personality traits in boys showing obesity of the mons-mammary-girdle type." The patients were all referred

because of difficulties in behavior only. They represent the group with the functional form of dyspituitarism.

In table 1 the physical findings in the 33 cases of the Fröhlich syndrome are presented. A history of developmental delay occurred in 3 cases.

The metabolic rate was measured in 16 cases. The range was from —16 to +31 per cent, with a median of —1 per cent; in most cases the results were within normal limits. Roentgenograms of the sella turcica were made in 12 cases, and the findings were all normal.

The distribution of the intelligence quotients is shown in table 2. The group had a high average rating, as determined by intelligence

TABLE 1.—*Physical Findings in 33 Boys with the Fröhlich Syndrome of Ages Ranging from 7 to 19 Years (Median 12 Years)*

	Number of Cases
Weights above the median for age and height (Baldwin-Wood tables)	
0 to 10 per cent	4
11 to 20 per cent	2
21 to 30 per cent	11
31 to 40 per cent	10
41 or higher	6
	33
Mons-mammary-girdle adiposity	33
Hypogenitalism	28
Small penis; normal testes	18
Small penis and testes	10
Tapering fingers	19
Smooth, velvety skin	14
Feminine appearance or mannerisms	7
Genu valgum	6
Miscellaneous	
Ocular findings; difficulty in convergence; slight limitation in visual fields	1
Chronic osteomyelitis	1
Old infantile paralysis	1
Choreiform movements	1
One or both testes undescended at first examination (descended six months later)	1
History of developmental delay	3

tests. Only 5 boys were below the average in intelligence, and 17 were in the superior group. This is a predominantly higher score than that for the entire series of patients in the Institute for Child Guidance, for which there is a median quotient of 99.

In the records of cases appended to the paper, besides data concerning the reference and the physical findings, all items in the record that can be characterized as aggressive or submissive are included. For example, in case 1, that of a boy aged 19 years, items considered as evidence of submissive behavior are the following: "The child was very bashful and sensitive . . . had a few companions, younger boys. In the family, as at school, he always tried to please; . . . was anxious to go on errands or to help in any way . . . was very

obedient . . . took good-naturedly his nickname 'Fatty' . . . had always tried hard in school, and, in spite of repeating many grades, was never truant or unwilling to go . . . had no girl friends. An older brother had taken a responsible attitude toward the patient, and he [patient] accepted the status of submissive follower and baby of the family." No items were found that could be listed as aggressive traits. The patient was characterized as submissive. In case 2, that of a boy aged 13 years and 9 months, a few items are listed under the head of aggression: "show-off behavior before adults" and "free and outspoken [manner] in interviews." Under the head of submission the following observations are included: He "was frankly afraid of boys because they teased him and was afraid to go to the toilet in school for this reason. He had fears [also] of the dark, storms and sickness. . . . He had a pleasant, happy disposition, . . . showed good deportment in school and was liked by teachers for this reason. In spite of diffi-

TABLE 2.—*Distribution of Intelligence Quotients (Stanford-Binet Intelligence Tests)**

Intelligence Quotients	Number of Cases
70- 79	1
80- 89	4
90- 99	6
100-109	5
110-119	5
120-129	8
130-139	1
140 +	1
No data	2

* The range was from 70 to 160, with a median of 110 to 119.

culty in school work he never was truant or presented a disciplinary problem. He passively accepted children's teasing and hitting . . . preferred friends younger than himself . . . admitted frankly his inferiority to others, that he was weaker and ugly, 'that even the littlest boy could run away from him' and that he could not defend himself because he was smaller and weaker." At 14 years he complained: "Boys don't ask me to play with them." "The mother told of efforts to make him more independent of her and to keep him from hanging around her all the time." The picture was that of a very immature and submissive boy.

In case 3 the weighting of the items is not easy. The problem fighting and bullying distinctly has to do with aggressive behavior. However, the bullying was only of little children. He preferred to play with children 2 or 3 years old. He was obedient at home and fond of helping with the housework. He frequently asked the teacher if he could help with chores. It is clear that the general picture was that of a submissive boy, whose aggression was expressed in a safe way. Aggres-

sive behavior with much younger children, such as younger siblings, is so common an item in all cases, including those in which the patient was characterized as submissive, that it is regarded as of minor importance in the weighting of aggressive tendencies.

In case 4, that of a boy aged 12 years and 6 months, the item "spoke freely and spontaneously in interviews" appears under the head of aggression. This manifestation also is common even in very submissive boys after the initial interview. In fact, a growing freedom in the interview with the psychiatrist characterized most submissive boys in the list. It is considered as evidence of aggression, though of minor importance. The same boy allowed his younger brother to impose on him. He was obedient at home and at school. He had few companions. Though he did poor school work, he was never truant and did not present a problem in deportment. He was regarded as sweet and passive. The items were prevailingly submissive.

An example of a very submissive boy, though one disobedient to his mother, is revealed in case 5. The patient raised his hand in class frequently, though often he did not know what the question was. The aggressive items have to do with disobedience and bids for attention. However, in the group he never joined in the games with others but stood around and watched. When he was hit and teased by other boys he never fought back. Though 11 years of age, he preferred to play only with a sister aged 5 years. Even in the interviews he had to be prodded and encouraged before he replied, because of timidity. He was characterized as submissive.

Enough data have been reported to indicate how the description of the personality as generally submissive or aggressive was determined. There was little difficulty in this connection with the group of patients with the Fröhlich syndrome, for the submissive behavior was marked. Of the series of 33 patients, 26 were easily characterized as submissive, 2 as displaying mixed behavior, with a fairly equal weighting in the two categories, and 5 as aggressive. This is an unusual finding, since, as already noted, the majority of children who came to the Institute for Child Guidance presented problems in aggressive behavior.

To aid in differentiating overtly aggressive and submissive behavior, the following criteria were established.

OVERT AGGRESSION AND SUBMISSION

Evidence of Aggression.

Problems in authority—rebellion and undisciplined behavior, i. e., active disobedience to teachers or other persons in authority, active violation of classroom discipline and assault reactions to children in the classroom.

Delinquency: truancy and stealing, except when proved to occur only as overtly submissive responses to another child or to other children.

Dominating behavior with other children, in the form of leadership and bullying.

Fighting with other children in defense or aggression.

Angry retorts and calling names, in response to nicknames or criticisms, with other children.

Joining readily in play with other children.

Disobedience to parents.

Fighting or quarreling with a sibling or siblings.

Evidence of Submission.

Good deportment in the classroom.

Obedience to teachers, parents and persons in authority.

Absence of truancy or classroom difficulties in behavior, in spite of retardation in school, poor school work or dislike of school.

Submission to other children and lack of friends because of fear of other children or timidity.

Fear of fighting.

Urging to join the play group required because of timidity.

Complacent acceptance of nicknames or insults, i. e., no fighting or angry retorts.

Obedience to parents; submission to infantilizing care or to the mother's refusal to take risks with the patient.

METHOD OF INVESTIGATION

The items were discussed with two students, and the plan was submitted to them. Student A wrote on summary sheets all items that could be considered under the category of aggressive or submissive behavior in 100 cases, taken seriatim from the files of the Institute for Child Guidance. Student B did likewise in another group of 100 cases. Each made independent scores for all the items gathered in the 200 cases and classified the behavior in each case as aggressive, submissive or mixed. The agreement in classification was 96 per cent. The same criteria were used to determine aggressive and submissive behavior in 1,000 children referred to the Institute for Child Guidance. Sixty per cent were classified as aggressive.[1] Correlations were then made between the classifications

1. As a by-product of this study, the manifestations of overt aggression are found to be classified on the basis of certain striking components: A. Dependence Aggression: The aggressive maneuvers are seen in early childhood as an attempt to compel the mother to remain in constant contact, to do things for the child and to protect it. Under this heading the stress may be on the contact, the infantile care or the protective phase of the maternal relationship.

B. Dominating Aggression: The activity has as its objective the control of the other person, originally to make the mother do whatever the child wishes. In

(Footnote continued on next page)

and the weight, height, age, intelligence quotient, ordinal position in the family and sex and the various problems which were reasons for reference. These particular studies are not included in this paper. It is worth noting from these studies, however, that the group of patients with the Fröhlich syndrome shows a higher proportion of instances of submissive behavior than any other.

Since the physical factors in the Fröhlich syndrome to which a psychologic response would be most likely are the obesity and the small genitalia, a study of the factors of aggression and submission was made on a series of male patients with varying degrees of obesity and on a small series of patients characterized by small genitalia. In the group of obese children and in that showing hypogenitalism, excluding patients with the Fröhlich syndrome, the same percentage of aggression occurred as in the total series of 598 patients; i. e., about 60 per cent showed aggressive behavior. The group of patients with hypogenitalism comprised 15 boys. Data on the group of obese male children are included in the table that follows.

TABLE 3.—*Relation of Weight to the Frequency of Aggression and Submission in a Group of 598 Problem Children* *

Weight Above or Below Medians for Age and Height, Percentage	Aggressive Behavior		Submissive Behavior		Mixed Behavior		Total
	No. of Cases	%	No. of Cases	%	No. of Cases	%	No. of Cases
—20 and below	4	66	1	17	1	17	6
—10 to —19	47	55	29	34	9	11	85
+ 9 to — 9	253	60	129	31	41	9	423
+10 to +19	38	61	17	27	7	12	62
+20 and above	13	59	8	36	1	5	22

* The range in age was from 2 to 18 years, and the median age, 10 years, calculated on the basis of the Wood-Baldwin tables.

contrast with dependence aggression, in which the child tries to compel the mother to treat him, even to dominate him, as in infancy, the objective is to compel the mother's obedience.

C. Negative Aggression: This includes certain forms of disobedience and negativistic and defiant behavior. The aggression operates only when the child is prevented from doing what he wishes. It blocks attempts to manipulate or modify the child's behavior, without necessarily involving initiative in aggressive behavior.

D. Exhibitionistic Aggression: Attention getting, showing off and clownish behavior come under this heading. Attention-getting behavior, however, may be a type of dependence aggression, such as the child's pulling at its mother's skirts for attention. Showing off may be in the form of real display ("See how wonderful I am"). On the other hand, in the form of clownish behavior the aggression may evoke attention by showing the child in a poor light, e.g., making a fool of himself in front of the group. This appears to be a type that occurs in generally submissive children, though it is a distinctly aggressive maneuver.

E. Sadistic Aggression: A special group must be assigned to children whose objective is not to lead the group or to dominate but to gain aggressive satisfaction by fighting, vanquishing or display of cruelty.

AGGRESSION AND SUBMISSION

In characterizing the children showing the Fröhlich syndrome as submissive, it is implied that, unlike most children referred to the Institute for Child Guidance as presenting behavior problems, they yielded fewer instances of behavior characterized by refusal to comply with the disciplinary requirements of the home, school or society at large; that is, they manifested relatively fewer instances of rebellious behavior. Furthermore, besides evidence of lack of rebellious behavior there appeared complaints of "shyness," "withdrawing behavior," "fear of fighting" and similar items illustrating withdrawal because of fear or anxiety from the ordinary social activities of children.

There is little difficulty in differentiating the terms aggressive and submissive when applied to studies of cases of overt behavior. For example, the boy (case 12), aged 15 years, who was referred because of truancy and disciplinary problems in the classroom, was described as generally defiant of authority. He was unpopular with boys because he was quarrelsome and fought and bullied. At home he insisted on special food and service and managed to get what he wanted. He tried to have everything his own way and to dominate every situation. He stole and played truant from school.

In the case cited the problems represent in each instance examples of aggressive behavior. The patient attempted to dominate and did not yield to the domination of others sufficiently for the purpose of adaptation to the ordinary requirements of parents, teachers or friends. The delinquent acts truancy and stealing are regarded also as evidence of aggressive behavior when they do not represent merely submission to a delinquent person or to a delinquent group. The social activities of the patient are easily characterized as examples of aggressive behavior, such as attempts to attack or to "set on others"—acts that stamp the original meaning of the word "aggress."

The first difficulty in applying the term is presented by children who do not actively rebel but refuse to yield to authority. A frequent complaint concerning a number of so-called lazy children, for example, is their resentment of persons interfering with their dawdling behavior. In such cases, when disobedience, defiance and the like occur, the behavior is characterized as aggressive. The acts in themselves must be so characterized, whatever the difference in the personalities that manifest them.

In 22 cases, for example, in which laziness was one of the reasons for reference, 17 children were predominantly aggressive and 5 submissive, according to the criteria already given. The term laziness was used to denote slovenliness, slowness, dawdling and general lack of energy (especially sleeping late) in 14 cases and to cover negativism or

disobedience, especially refusal to work or study or help with housework, in 5 cases. The definition was not clear in 3 instances.

In characterizing behavior as generally aggressive, difficulty arises when the aggression occurs in one or more of the several social relationships of the child but not in all. Aggressive behavior in the form of quarreling and teasing seems to occur most frequently in relation to siblings. Disobedience to a parent is more frequent than disobedience to a teacher. Expression of aggressive behavior in children occurs more frequently when they play with younger than when they are with older children. On that basis one would infer that ordinarily a disciplinary problem in the classroom is greater evidence of aggression than a problem in rivalry between siblings.

In each situation there is, furthermore, a variation in the expression of the aggressive maneuver. Such differences are revealed clearly in the anamnesis when the aggression creates a problem in social adjustment. Ordinary teasing and occasional fighting among brothers occur too frequently in family life to be considered a special problem. When sibling rivalry remains at a level of savage attacks and constant domination, a problem in aggressive behavior is manifest. In the disciplinary problems in the classroom likewise there is a wide variation, starting with numerous forms of attention-getting behavior and ending with open defiance of every rule.

It is premature to attempt a refined measure of aggressive behavior at the present stage of this study. The patients were characterized as aggressive only when the data were predominantly significant of that type of behavior. In each case the data are recorded under aggression and submission, and the reader may judge the ease of differentiation. When doubt exists as to either category because there appears a fair balance of data under both heads, the data are characterized as mixed.[2]

The clearest examples of problems in submissive behavior are shown by children who yield readily to the domination of others and typically do not utilize the aggressive maneuvers described. They are characterized as submissive because of absence of aggression and evidence of compliance. In many situations compliance is inferred because a problem in aggression is lacking. Retardation in school presents a situation that calls typically for aggressive behavior in the form of refusal to go to school, truancy or rebellious behavior in the classroom. The child left back in the grades because of failure has the problem of adaptation to work in which there is little or no satisfaction, often to

2. A description of the range of aggressive behavior from primitive hostility to indirect, highly modified hostility has been made in a previous study (Levy, D. M.: Use of Play Technic as Experimental Procedure, Am. J. Orthopsychiat. **3**:266 [July] 1933; Hostility Patterns in Sibling Rivalry Experiments, ibid. **6**:183 [April] 1936).

the taunts of family and schoolmates and to a lowered self-esteem. The problem is analogous to that of the adult who is compelled to work daily at a job that is dull, monotonous and difficult and who must suffer at the same time the gibes of his fellow-workers. One would expect, especially in a child, with his less disciplined personality, overtly expressed rebellion against such conditions, or at least a period in which rebellion is manifest before the period of submissive adaptation. In the first case of the Fröhlich syndrome in my series, the patient, in spite of repeating many grades, was never truant or unwilling to go to school. Consistent with this attitude was his general submissive relationship to others. At home he was very obedient and anxious to go on errands and please every one. He accepted his status in the family as a submissive follower of an older brother. With schoolmates he was shy and withdrawn. At 19 he had a few companions among younger boys, but no girl friends.

When problems in social relationships are characterized predominantly by submissive or aggressive behavior the lines of demarcation are easily drawn. In normal behavior the mixture becomes more perplexing. Nevertheless, the terms aggressive and submissive are used to characterize presumably normal social adjustment, in the sense of assertiveness and compliance. In current literature one now reads of healthy (i. e., "normal") aggression and submission. The problem of expressing "instinctive" aggressive and submissive tendencies has been shown, especially through psychoanalytic investigations, to be a fundamental adaptation in the life of every person. When used to express normal adaptive processes the terms lose their usual connotations of contentiousness and servility (or "resignation"). Normal aggression implies behavior in which the person asserts such independence of speech or action in any given social situation as is asserted by presumably normal persons under similar circumstances. On the same basis, normal submission implies compliance in social situations according to norms of compliant behavior. Though admittedly an unsatisfactory definition, it represents the way one actually determines such behavior. A child who is occasionally disobedient, who protests against an early bedtime or who teases a brother or sister may display in all these situations aggressive behavior typical of most children. On the other hand, a child who is always obedient at home and perfect in conduct at school and who never quarrels or fights with companions is unusually compliant. In drawing such conclusions, one assumes on the basis of personal and clinical experience a norm for aggressive and submissive behavior. This assumed norm applies also to considerations of age, sex and various social and cultural milieus. A girl who freely expresses her opinions before her elders may have manifested a commonplace and accepted bit of aggression in 1930 but unusual boldness

in 1890, as measured by typical modes of expression within cultural groups. For such behavior statistical norms might be established. In fact, certain frequencies that apply to this problem have been worked out; e. g., frequencies of dawdling, refusal of food, temper tantrums and hostile expressions in sibling rivalry.

The terms assertive and compliant are probably to be preferred in describing behavior in ordinary social relations. The terms aggressive and submissive might then represent exaggerated forms of assertion and compliance. In infancy these personality attributes are manifested in primitive, i. e., unmodified, forms. It is part of the problem of social adaptation to modify them and a frequent (essentially the important) problem of child guidance to aid in this task. In utilizing the terms submission and aggression rather than those of assertion and compliance, there is the advantage of using terms for behavior that represent the same dynamics, varying only in a quantitative way.

Of the words used as synonyms for, or as part of the concept of, aggression, the commonest are domination, energy, initiative and masculinity. Domination in the form of bullying or of leadership is easily comprehended as evidence of aggressive behavior. But domination through dependent behavior or utilization of illness or fear or even kindness, is certainly not evidence of overt aggression; that is, though aggressive behavior is used as a means of social domination, submissive behavior may be used likewise.[3]

Initiative in making friends or in looking for a job is given as an illustration of aggressive behavior. The term initiative is used to denote activation of social enterprise by the person himself, i. e., with little or no pressure from others. But initiative in itself does not imply aggression. A person may, for example, consistently initiate social contacts in which he plays a submissive rôle, as in the case of a child who always started a game in which he played the part of follower. Initiative in getting a job may be evidence of obedience to the mother, of fear of the father, of a responsible attitude and the like, in a generally submissive person. Conversely, a very aggressive child, in the sense of defying authority and bullying his companions, may show little initiative. Whatever the theoretical explanation of "self-starting" energy, it does not involve necessarily aggressive behavior. The submissive as well as the aggressive maneuver may manifest domination and initiative.

3. A child, like an adult, may be placed in a position of leadership or domination through superior knowledge, ability or favor; e. g., the child may dominate through possession of toys or special skill in baseball, or deference may be made to his judgment because of his superior knowledge of a game. In such cases leadership has not come about through aggression, though such experience may favor its development.

The word energetic is often used as a synonym for aggressive. There is implied in this usage the energy theory of aggression, yet the aggressive act does not necessarily involve more expenditure of energy than the submissive one. Children who are referred with the complaints "restless behavior," "constantly on the go," "overactive," etc.—traits usually listed under the term hyperkinetic—demonstrate, however, a high degree of aggressive behavior. A special study of 25 cases of hyperkinesis in children, for example, as compared with 15 cases of the Fröhlich syndrome, shows three or more times as frequently the following complaints: destructive behavior, disobedience, temper tantrums and fighting. In further marked contrast, the group of children with the Fröhlich syndrome presented three or more times as frequently the following complaints: fears, day-dreaming, laziness and timidity.[4] It might seem, therefore, that one is comparing groups in which differences in aggression are simply differences in the manifestation of energy.

A number of problems arise out of these findings that, unfortunately, cannot be solved by available data. It is not yet possible to differentiate the children in the group with hyperkinesis whose energy output is physiologically greater than normal, who are, in other words, more naturally active children, from those whose increased energy represents a restless acting out of emotional difficulties. Furthermore, a number of hyperkinetic children are cured of the so-called hyperkinesis when their problems in social adjustment are solved (especially by removal from a difficult home situation to affectionate relatives or other adults).

The group of hyperkinetic children are relatively young as compared with the group showing Fröhlich's syndrome. Just over half are 6 years of age or younger, whereas the children of the latter group are mostly over the age of 9 years. Children under 6 years of age yielded the highest proportion of problems in aggression (in the lists of the Institute for Child Guidance), regardless of the presence of hyperkinesis.[5] This finding is usually explained as the typical behavior of children, who are more likely to act out their difficulties (especially in the form of temper tantrums, destructive behavior and fighting) because of their still unmodified primitive aggression. Whatever the reason for the high proportion of aggression in the lists of the younger children, the theory that differences in aggression depend on differences in energy is not helped by the data from this clinic. Nor can the fact that the groups of younger children show more problems in aggression than

4. The cases of the Fröhlich syndrome were compared with those studied by E. Ginsburg (A Study of the Factors Associated with Variations in the Intensity of Hyperkinesis in Children, Smith College Thesis, 1932).

5. Study on the correlation of age and aggressive-submissive behavior in 1,000 cases at the Institute for Child Guidance already mentioned.

the group of older children with the Fröhlich syndrome be explained by the factor of age, since the latter had maintained their generally submissive pattern since infancy.

Aggression is often assumed to be a prerogative of the male sex and is then used as a synonym for masculinity. The assumption usually extends to the theory that males are instinctively aggressive and females submissive—at least, that there is a marked quantitative variation of these "instincts" in the sexes. The terms sadistic and masochistic are used in a similar way. Such assumed differences in instinctive make-up are derived chiefly from the mating act, the fighting propensities of males and the maternal functions. From the sexual act, especially, the important psychologic contrasts for the sexes have been drawn—the male, active, aggressive, commanding, on top, penetrating, sadistic; the female, passive, submissive, obedient, on bottom, receptive, masochistic. Strength and sexual function favor the aggression of males. Menstruation, pregnancy, childbirth and lactation are stock examples of the protective needs of females and, hence, of their natural social adaptation in the form of feminine, submissive behavior.

The differences in the behavior of the sexes may be evaluated in various ways. The female may be regarded as the aggressor, utilizing the male as a submissive agent to her sexual needs, in spite of his apparently aggressive maneuvers. Viewed differently, both the male and the female are regarded as passive agents of instinctive forces, to which their particular performances are merely necessary details of adaptation. Again, sexual behavior, viewed nonbiologically, is regarded as a natural exchange of pleasure, in which neither is victim nor victor, aggressor or submitter, but both are active and volitional partners.

There are obviously various factors in sexual behavior besides aggressive and submissive elements. However, within the framework of the act there is a necessary aggressive adaptation for the male and a submissive adaptation for the female. Difficulties in these relationships are found typically in the neuroses. They represent the commonest problems in psychic impotence and frigidity. The pattern of aggression as a particularly necessary male element in coitus is obvious, whatever the attitudes of the partners and regardless of the sex of the partner who initiates the act.

The problem of acceptance by the female of male aggression extends, of course, beyond the sexual performance into various phases of family and general social life. This adaptation is by no means a simple submitting to the aggressor. Without elaborating the problem, the point should be emphasized that in utilizing the terms masculine and feminine as synonymous for aggressive and submissive a distortion results in the meanings of many social situations. Maternal behavior, for example,

is distinctly aggressive. Aggressive as well as submissive adaptations are essential processes in all the social relationships of women, and their variations in this regard are as manifold as those of the opposite sex. In trying to apply the terms to our groups, it was found that the words masculine and feminine were more restricted and forced than the terms aggressive and submissive.

PSYCHIC AND SOCIAL AGGRESSION

In this paper I have dealt with aggression and submission in terms of overt behavior. Presumably, a child may be aggressive in his feeling and attitude, yet outwardly submissive. On the other hand, a child may be outwardly aggressive in spite of strong submissive tendencies. In the first case it is usually inferred that the aggression of the child is blocked by fear of the consequences of his aggression; in the latter, that the aggressive behavior is compensatory or represents a reaction against a submissive tendency. One might, therefore, differentiate between psychic and social aggression. The argument, applied to the Fröhlich syndrome, would stress the point that there have been studied only the overt manifestations of this behavior, without any knowledge of the real or true determinants. This does not affect the finding that patients with the Fröhlich syndrome are as a group characterized by a remarkably submissive adaptation to social life. The question remains: How is this submissive adaptation determined? That it cannot be explained in terms of lessened energy or obesity or hypogenitalism has already been shown. It is possible, of course, that all the submissive subjects of the Fröhlich syndrome may be aggressive personalities that are blocked in their aggressive behavior. It would be a remarkable coincidence that a selection of patients on the basis of a certain type of obesity happened to include so large a group manifesting neurotically determined submissive behavior. In the data on the group there is an abundance of complaints of fear. It is chiefly in the form of withdrawing behavior, shyness, timidity and the like. One may infer, on the one hand, that the problems of fear arise out of a submissive tendency, a fear of the aggression of others, or, on the other hand, that the submissive behavior is a derivative of fear. There is no way of determining directly the original endowment of aggression or submission. One can measure only in manifestations of social behavior or in the fantasy life. Since no psychoanalytic studies have been made of these patients, one does not have the advantage of material from dreams or free associations. However, if the dream material should show tremendous evidence of aggressive behavior the problem as stated would remain; namely, is the prevailing submissive tendency primary or secondary? One may work on the assumption that in the study of a group of patients

the outward manifestations of behavior follow natural trends. In other words, a "naturally" submissive child would take a submissive direction unless the aggressive tendency was unusually magnified by elements in the life experience. On that basis one would assume that patients with the Fröhlich syndrome showing predominantly aggressive behavior would, in contrast with the others, reveal evidence in early life of strongly determining environmental factors. In the few cases of unquestionably aggressive behavior this was, indeed, the case. Of the 33 patients, 5 were characterized as aggressive, and of these, 3 were predominantly so.

One of the very aggressive persons (case 12) was a boy aged 15. He was overprotected for four years as an only boy and was breast fed for fifteen months and then markedly rejected when another baby came. He had been a large eater since infancy and had always demanded special dishes. There was an overt hostility to the brother and to the father. The dreams were full of sadistic attacks and breast fantasies. Of the features especially powerful in evoking hostile behavior the most potent was the experience of rejection following a period of overprotection. There is no other instance like it in the entire series. The rivalry with the brother and father was an added factor. The patient in infancy was "a very good baby." The difficulty had its onset with a change in maternal attitude after the birth of the younger brother. The items listed under aggression were bullying, fighting, truancy, stealing and general defiance of authority; the patient "insisted that he was always right and wanted every one to go his way;" he tried to dominate every situation, made special demands for himself at home, special service and special foods, and got what he wanted.

The second patient characterized as very aggressive (case 25) was a boy aged 9 years and 9 months. In his life history there were also conditions unusually favorable to the development of a powerful aggression. In his first three years he lived with a childless aunt, who strongly indulged him. This aunt was psychotic and gave him money to kill his mother. The father, who had dementia paralytica, was at a state hospital for a year while the boy lived with his own mother. He lived in a very bad neighborhood and had a number of delinquent companions. The patient bullied other children, stole and was truant; he fought with his brother and other children and assumed a dominating position in the home; nevertheless, he was a "model boy" in school.

In the third instance of very aggressive behavior, the data for the early family life were inadequate (case 15). In the remaining two patients in the aggressive group, the aggression was not especially pronounced, though the items favored that classification.

A study of the factors in the early background in the entire series of cases shows no evidence that all the experiential items were stacked in favor of submissive behavior. There were a number of instances of strong overprotection. However, the response to overprotection may be aggressive or submissive. Several instances occur of long nursing care because of illness. Of these the best is case 28, in which there were long periods of hospitalization and the mother used the patient's ill health as a proof of the need of her protective care. This has been shown in a previous study to evoke aggressive as well as submissive behavior.[6]

CONCLUSIONS

One may conclude, therefore, that the submissive behavior in the cases of the Fröhlich syndrome studied was due to a constitutional factor. It cannot be explained by the early life history, by a psychologic response to the obesity or the hypogenitalism or by the maternal attitude. Aggressive and submissive tendencies are presumably based on instinctive responses in social life. The strength of these responses is determined both by constitutional and by experiential factors. On the basis of this study I should conclude in general that, though constitutionally determined, a trend may be magnified or diminished by the life experience.

SUMMARY

The frequent observation of submissive behavior in boys with the Fröhlich syndrome was tested in 33 cases. The majority were found to be submissive, in contrast to the prevailing finding of aggression in 1,000 children referred to the Institute for Child Guidance.

An attempt was made to define the terms and to establish criteria for aggressive and submissive behavior. The criteria were then applied to various groups of children, in order to determine the genesis of submissive behavior in the group of boys with the Fröhlich syndrome. The possibility that submission represents an adaptation to certain features of the syndrome, for example, small genitals, obesity or lessened total energy, was studied by applying the criteria for aggressive and submissive behavior to a group of children with small genitals who did not show the Fröhlich syndrome, to various groups arranged according to weight in relation to age and height and to a group of children with hyperkinesis. These studies showed that the submissive behavior in the group with the adiposogenital syndrome was not explainable as a response to any one of these particular factors.

6. Levy, David M.: Maternal Overprotection: A Human Relationship Study, New York, W. W. Norton & Company, to be published.

A study was then made of the life histories to determine whether the early experiences, including the maternal attitude, favored a submissive adaptation. Except in a few cases, in which the early experience was unusually favorable to aggressive behavior, the social factors in the background of patients with the Fröhlich syndrome were not in significant contrast with the social factors for the group used as a check. Since the submissive behavior could not be derived from adaptive response to a particular factor in the syndrome or to any special pattern of social experience, the conclusion was drawn that it is determined by the endocrine factors basic to the syndrome, i. e., that it represents a constitutional response. That the aggressive response in a case of the Fröhlich syndrome can be magnified by social factors was indicated in a few instances, demonstrating that in special types of environment submissive factors can be overweighted by aggressive factors, regardless of the constitutional tendency.

REPORT OF CASES

CASE 1.—A youth aged 19 years, the fifth of five children, who had reached the first year of high school and had an intelligence quotient of 74, was referred because of retardation in school and withdrawing behavior.

Physical Findings.—The height was 4 feet and 10 inches (147.3 cm.), and the weight, 145 pounds (65.8 Kg.) (40 per cent above the median). There was girdle adiposity. The penis was small (½ inch [1.27 cm.]), and there was a female distribution of pubic hair. The fingers were tapering. The developmental history showed general delay. The right leg was 6 inches (15 cm.) shorter than the left, with immobility of the right knee. An operation had been performed for osteomyelitis, with excision of the head of the femur, at the age of 7 years.

Evidence of Submission.—The child was very bashful and sensitive. He had a few companions, among younger boys. In the family, as at school, he always tried to please; he was most anxious to go on errands or to help in any way. He was very obedient. He took good-naturedly his nickname "Fatty." He had always tried hard in school and, in spite of repeating many grades, was never truant or unwilling to go. He had no girl friends. An older brother had taken a responsible attitude toward the patient, and the latter accepted the status of submissive follower and baby of the family.

Evidence of Aggression.—None was present.

Characterization.—The patient was characterized as submissive.

CASE 2.—A boy aged 13 years and 9 months, the younger of two children, who was in grade 6 B at school and had an intelligence quotient of 83, was referred because of retardation in school and rolling his head from side to side.

Physical Findings.—The height was 57.2 inches (144.8 cm.) and the weight 103.5 pounds (46.9 Kg.) (24 per cent above the median). The skin was smooth and velvety. The hair was fine and silky; there was no axillary hair and very scanty pubic hair. There were girdle adiposity and gynecomastia. The penis was short (length 3.5 cm.), and the testes were normal. The fingers were long and tapering. There was no evidence of rolling of the head. The visual fields

and optic disks were normal. There had been developmental delay; the patient sat up at 11 months, crept at 14 months, walked at 20 months and talked at 3 years and 6 months. When he was examined at the age of 14 years and 9 months (no medication had been employed), the height was 61.8 inches (156.9 cm.), the weight 113 pounds (51.3 Kg.) and the length of the penis 7 cm. There was a female distribution of pubic hair, which was normal in amount.

Laboratory Tests.—The basal metabolic rate was + 21 per cent. When the test was repeated a week later it was + 25 per cent. A roentgenogram of the sella revealed normal dimensions (the anteroposterior diameter was 0.9 cm., and the supero-inferior diameter, 0.7 cm.). The blood sugar value was normal (101 mg. per hundred cubic centimeters).

Evidence of Submission.—The patient was frankly afraid of boys because they teased him and was afraid to go to the toilet in school for this reason. He had many fears—of the dark, storms and sickness. (In infancy he had been very inactive and "appeared to be doped.") He had a pleasant, happy disposition. He showed good deportment in school and was liked by teachers for this reason. In spite of difficulty in school work, he never was truant or presented a disciplinary problem. He passively accepted children's teasing and hitting. He preferred friends younger than himself. He grinned constantly. He frankly admitted his inferiority to others, that he was weaker and ugly, that "even the littlest boy could run away from him" and that he could not defend himself because he was smaller and weaker. He complained that boys did not ask him to play with them (his age was 14). The mother told of efforts to make him more independent of her and to keep him from hanging around her all the time (at the age of 14). He had no interest in sports.

Evidence of Aggression.—He displayed show-off behavior before adults and was free and outspoken in interviews.

Characterization.—The picture was one of an immature, submissive, introverted boy. He was characterized as submissive.

CASE 3.—A boy aged 10 years and 4 months, the eldest of three children, who was in grade 3 at school and had an intelligence quotient of 96, was referred because of fighting and bullying.

Physical Findings.—The height was 55.5 inches (140.97 cm.) and the weight 98.5 pounds (44.7 Kg.) (34 per cent above the median). There was girdle adiposity. The penis was very small. Genu valgum was present. The patient had tapering fingers and soft, smooth skin and was considered "effeminate looking."

Evidence of Submission.—He showed preference for playing with children of 2 and 3 years of age, until the father stopped it when he was at the age of 10 years. He was obedient and fond of helping with the housework at home and asked the teacher if he could help with chores.

Evidence of Aggression.—He bullied and fought with younger boys. He was active in sports and free and forward in interviews, boasting of his strength and fighting ability.

Characterization.—The patient was characterized as submissive.

CASE 4.—A boy aged 12 years and 6 months, the elder of two children, who was in grade 7 A at school and had an intelligence quotient of 103, was referred because he did poor school work and was "lazy and lethargic."

Physical Findings.—The height was 60.6 inches (153.9 cm.) and the weight 121.5 pounds (55.1 Kg.) (25 per cent above the median). There was girdle adiposity. The penis was small, but the testes were normal. The skin was soft and smooth. (At the age of 13 years and 1 month the height was 62.1 inches [157.7 cm.] and

the weight 129.5 pounds [58.8 Kg.].) The mother had noticed the small size of the penis.

Laboratory Tests.—The basal metabolic rate was —5.

Evidence of Submission.—The patient allowed his younger brother, aged 10 years, to impose on him. Except that he lacked initiative and was slow, for which he was nagged, he was obedient at home and in school. He had few companions, preferred younger boys and showed no initiative in making friends. He was "sweet and passive" and complacent about school.

Evidence of Aggression.—He spoke freely and spontaneously in interviews. He was active in sports.

Characterization.—The patient was characterized as submissive.

CASE 5.—A boy aged 11 years and 1 month, the second of three children, who was in grade 6 B at school and had an intelligence quotient of 80, was referred because of "queer behavior." The teachers had noticed a "continuous grin" and an "inability to express himself."

Physical Findings.—At the age of 10 years the height was 57 inches (144.78 cm.) and the weight 105 pounds (47.6 Kg.) (31 per cent above the median). He had a feminine physical make-up. There were girdle obesity and marked gynecomastia. The genitals were small. The fingers were tapering. Genu valgum was present. At the age of 11 years and 1 month the height was 58 inches (147.3 cm.) and the weight 114 pounds (51.7 Kg.).

Laboratory Tests.—The metabolic rate was —6 per cent. A roentgenogram of the sella showed it to be normal, except that it was large for a child of his age. Tests for sugar tolerance and a blood sugar determination gave normal results.

Evidence of Submission.—The patient did not join in games with others but stood around and watched. In one neighborhood he was hit and teased by other boys, without fighting back. In his present neighborhood the boys were "kind" to him. In interviews he had to be prodded and encouraged before he would reply, because of timidity. He lacked initiative and much preferred playing with a 5 year old sister. His "temper tantrums" consisted chiefly of crying when scolded.

Evidence of Aggression.—He constantly raised his hand in class, though he often did not know what the question was. When asked he grinned. He was disobedient to his mother.

Characterization.—He was characterized as submissive.

CASE 6.—A boy aged 13 years, an only child, with an intelligence quotient of 113, was referred because of failure in school, lack of initiative and interest and overdependence.

Physical Findings.—The height was 57.2 inches (140.2 cm.) and the weight 113.7 pounds (51.7 Kg.) (34 per cent above the median). There was a girdle distribution of fat, and the penis and testes were small. The fingers were tapering. There were marked limitation of movement of the right lower extremity and weakness of the right hand. The patient was able to walk with a limp and could use the right hand for dressing and eating but not for writing. He had had infantile paralysis at the age of 7 years.

Evidence of Submission.—He insisted that his mother should accompany him to school. He was described by a family friend as "a charming boy, who ought to have been born a girl." He was always obedient at home and at school, and friendly and good natured. He had to be prodded in every classroom because of

listlessness. He accepted his paralysis good-naturedly and was complacent. When with his mother in a restaurant, he accepted her infantilizing care, letting her feed him. In interviews his responses were childish and immature. (His lack of interest in sports was explained by the physical handicap of paralysis.)

Evidence of Aggression.—He was spontaneous and free in interviews.

Characterization.—He was characterized as submissive.

CASE 7.—A boy aged 12 years and 8 months, the second of five children, who was in grade 7 B at school and had an intelligence quotient of 110, was referred because of overweight, an abnormally large appetite and fear of other children.

Physical Findings.—The height was 63 inches (160 cm.), 5 per cent above the median, and the weight 167 pounds (75.7 Kg.) (56 per cent above the median). There was girdle obesity, with gynecomastia. The penis was short, but the testes were normal.

Laboratory Tests.—The metabolic rate was 31 per cent. The roentgenogram of the sella revealed normal structure (size 1 by 1 cm.). The blood sugar content was 104 mg. per hundred cubic centimeters.

Evidence of Submission.—The patient was "afraid to play with boys because they fought with him, and he was not able to fight back." The mother urged him to fight back. He stayed with his mother constantly and was with her wherever she went. (He was "a good baby.") He was always quiet and never gave any trouble in school. He accepted his nickname "Fat" good-naturedly. In interviews he was submissive and compliant. He annoyed his mother by his constant nagging and staying in the house.

Evidence of Aggression.—When his mother refused him food he "hollered at her." He liked sports and was active in swimming class. He teased and fought with younger siblings.

Characterization.—He was characterized as submissive.

CASE 8.—A boy aged 12 years and 7 months, the eldest of three children, who was in grade 7 B at school and had an intelligence quotient of 123, was referred because of laziness and failure in school work.

Physical Findings.—The height was 60.2 inches (159.9 cm.) and the weight 114 pounds (51.7 Kg.) (24 per cent above the median). There was girdle distribution of fat. The genitalia were small, the testes unusually so (size of a pea). The mother said she had noticed that her younger boy, aged 5 years, had larger genitals than the patient. There were difficulty in convergence with either eye and slight limitation of the temporal field of vision on the perimeter. The patient was handicapped in sports because of his having to wear glasses and his slowness. Myopia was present. At the age of 14 years the height was 63 inches (160 cm.) and the weight 130 pounds (59 Kg.).

Laboratory Tests.—A roentgenogram showed the sella turcica to be very small. The basal metabolic rate was —10 per cent. At the age of 14 years and 2 months it was —13 per cent.

Treatment.—Anterior pituitary extract, in doses of from 15 to 20 grains (972 to 1,296 mg.) daily, was consistently taken by mouth for four months.

Evidence of Submission.—The patient was sensitive, and his feelings were easily hurt. When the boys made fun of him he became hurt and refused to play. He cried readily. When a boy wanted to fight him he often replied: "My mother doesn't want me to fight." However, if made to fight he held his own. He got his way with his mother by weeping. He was reported to have no companions at school and never brought a boy to his home. In an interview, when asked how

he got along with his companions, he replied: "They let me play baseball sometimes." He showed no rebellion to his nickname "Fat." With playmates he did "what they let him do and not what he forced them to let him do." He frankly said that he wanted to remain a child and not grow up, so that he could always be at home with his parents and not have to work. A typical question: "Will I be sent to camp this summer?" showed that he tacitly accepted the situation and asked in the submissive form. He displayed complacent, infantile, dependent behavior.

Evidence of Aggression.—He quarreled and fought with his sister. There was a change from evasive to outspoken and impertinent behavior in the first three psychiatric interviews.

Characterization.—He was characterized as submissive.

CASE 9.—A boy aged 11 years and 9 months, the younger of two children, who was in grade 4 at school and had an intelligence quotient of 127, was referred because of "nervous tremors and twitching of the body" and stuttering.

Physical Findings.—The height was 55 inches (137.7 cm.) and the weight 90 pounds (40.8 Kg.) (21 per cent above the median). At the age of 13 years and 10 months, the height was 59 inches (149.86 cm.) and the weight 103 pounds (46.7 Kg.) (14 per cent above the median). There were girdle adiposity and female distribution of fat. The skin was soft and smooth. The penis was small, and the testes were normal. Genu valgum was present. Choreiform movements (sequelae to acute chorea [?]) of the arm were present at the first and not at the second examination. The patient was effeminate in appearance.

Laboratory Tests.—The metabolic rate was —11.

Evidence of Submission.—The boy was obedient to a dominating father, of whom he was afraid and in whose presence the stuttering increased. Once he lost the money for groceries and returned home and locked himself in a closet.

Evidence of Aggression.—He mixed well with other boys, was elected president of his class and was the leader of his group at camp, in spite of his stammering. On his own initiative, he studied to get into an advanced class.

Characterization.—The patient was characterized as aggressive.

CASE 10.—A boy of 9 years and 11 months, an only child, who was in grade 5 A at school and had an intelligence quotient of 125, was referred because of his inability to get along with other children.

Physical Findings.—The height was 57.5 inches (146 cm.) and the weight 106.5 pounds (48.3 Kg.) (27 per cent above the median). Girdle adiposity was present. The fingers were tapering. Both the penis and the testes were small. He was reported to have been "restless since infancy and rather clumsy." He had been prematurely born, at 8 months. There had been no developmental delay except in regard to teething (at 14 months). Restless activity had been noted since the age of 1 month. At 3 years of age he always entered a room "with a burst and a bound."

Laboratory Tests.—The metabolic rate was —10 per cent. The roentgenogram of the sella revealed normal structure. At the age of 11 years and 4 months the metabolic rate was —7 per cent.

Treatment.—There were further examinations at the ages of 10 years and 3 months, 10 years and 5 months, 10 years and 6 months, 10 years and 7 months, and 11 years and 6 months, and frequently thereafter. At the last examination, at 11 years and 7 months, the height was 61.5 inches (156 cm.) and the weight 131 pounds (59.4 Kg.). There was no noticeable change in the relative obesity

or the size of the penis after the boy had taken anterior pituitary extract by mouth for fourteen months in doses of 15 grains (972 mg.) a day, increased to 25 grains (1,619 mg.) and then to 30 grains (1,943 mg.); then 5 cc. of solution of anterior pituitary was administered daily intranasally for eight months, and 1 grain (64.7 mg.) of thyroid extract daily for three months, accompanied by diet and exercise.

Evidence of Submission.—The patient was afraid to try various sports and was timid, for instance, in learning to roller-skate or swim. He had to go twice to a physician before he could be induced to open his mouth for an examination of the throat (at the age of 8 years), and a similar reluctance was noted with the dentist. He was obedient to his parents, who felt that he presented no problem at home. He liked to tease or pretend to slap a boy and then run away. He was afraid to fight.

Evidence of Aggression.—He got along well with a playmate and had no special difficulty with other children until the age of 6 years, when (and frequently afterward) the school reported difficulty with his teasing other children. There were frequent complaints also from neighbors that he annoyed other children. The children avoided him, and at camp or on the school playground he stood around looking on. "His attitude was one of being content if he was not made to go out and play with other children." Examples of his teasing were putting tacks on chairs and tripping children. His behavior was described chiefly as that of a pest. He did not fight. He had had temper tantrums in early childhood, in which he hit or threw things at other children. He displayed show-off behavior, e.g., making faces in class. He showed initiative in selling magazines. He was outspoken and showed off in interviews. In the reception room he was observed to walk slowly up to a very small girl, lunge at her suddenly and then laugh at her fright.

Characterization.—His behavior was characterized as mixed.

CASE 11.—A boy of 12 years, the youngest of four children, who was in grade 6 B at school and had an intelligence quotient of 126, was referred because of truancy, temper tantrums and timidity.

Physical Findings.—The height was 57 inches (144.7 cm.) and the weight 126 pounds (57.2 Kg.) (55 per cent above the median). The skin was smooth and velvety. There was girdle adiposity with gynecomastia. The penis was very small (2 cm. in length), as were the testes. On examination at the age of 12 years and 11 months, the length of the penis, after the pubic fat was pushed back, was from 2½ to 3 inches (6 to 7.5 cm.); the testes were normal. There had been no developmental delay. The fingers were tapering. The patient appeared listless and showed lack of energy. The diagnosis was hypopituitarism and hypogonadism.

Treatment.—The boy was sent to a clinic for the treatment of endocrine disturbances.

Evidence of Submission.—He was timid and shy with strangers and ill at ease in their presence and had nothing to say. He was afraid of the dark and would not go into a dark room in his own home; he was also afraid to go out alone at night. When his father denied him a privilege he "just went into the next room and cried bitterly." He was obedient to the father. In infancy and early childhood "he was not aggressive. He always feared the dark or being left alone. He was up all night if his mother didn't kiss him good night." Her best weapon in punishment was to refuse this good night kiss. The school reported that his conduct was always good. He was affectionate, unselfish and obliging and always ready to do errands for any one and to share his possessions. He

was a favorite with principals and teachers, who saw no reason for his being referred. He had no companions until he was 10 years old and until then was timid with boys.

Evidence of Aggression.—In infancy he was always stubborn, always wanted his own way and usually got it. He was disobedient to the mother and yelled at her. Solitary stealing and truancy occurred in the past two years. He was one of a gang of five boys.

Characterization.—He was characterized as submissive.

CASE 12.—A boy of 15 years, the second of three children, who was in grade 8 B at school and had an intelligence quotient of 125, was referred because he was truant and presented a disciplinary problem in the classroom.

Physical Findings.—The height was 67.8 inches (172 cm.) and the weight 190 pounds (4.1 Kg.) (42 per cent above the median). There was girdle adiposity. The penis was short; the testes were normal. The fingers were tapering. Genu valgum was present. The patient was considered "effeminate."

Laboratory Tests.—A roentgenogram of the sella revealed normal structure; the anteroposterior diameter was 1 cm. and the height 0.9 cm.

Evidence of Submission.—The patient preferred to play with younger boys. In infancy he had been "a very good baby."

Evidence of Aggression.—He was unpopular with boys because of his bullying and fighting. He stole, was truant and was generally defiant of authority. He "insisted that he was always right and wanted every one to go his way." He tried to dominate every situation. He made special demands for himself at home, e. g., special service at the table, and got what he wanted. He was active in swimming and hiking, liked camp and initiated projects for himself.

Characterization.—He was characterized as aggressive.

CASE 13.—A boy aged 17 years and 3 months, the youngest of four children, who was in grade 8 B at school and had a low average intelligence, as shown by the army alpha test, was referred because of retardation in school. Vocational guidance was also desired.

Physical Findings.—The height was 63 inches (160 cm.) and the weight 161.5 pounds (73.25 Kg.) (36 per cent above the median). Girdle adiposity and gynecomastia were present. There were scanty axillary hair and a moderate amount of pubic hair of female distribution. The fingers were tapering. The penis was small, and the testes were normal. The legs were short. There was a history of developmental delay (he talked at 2 years and walked at 2½ years).

Laboratory Tests.—The metabolic rate at the age of 10 years was —17 per cent. The sella turcica showed widening and absence of the posterior clinoid processes.

Treatment.—The patient had received pituitary treatment since early childhood. At the time of the examination he was taking daily 6 grains (388 mg.) of mixed thyroid and pituitary extract by mouth. He had received thyroid treatment since the age of 14 months. The diagnosis of hypopituitarism and dyspituitarism was made in early childhood.

Evidence of Submission.—He was apologetic and self-deprecatory during the physical examination. He had a quiet, pleasant disposition and was easily led. He had few friends. In the first year of life he was inert and had always to be prodded to prevent his falling asleep at the breast. The mother said that the child slept more than her other children and was less active. He yielded to the mother's bathing him until the age of 15 years. He accepted remarks about his

stupidity complacently. In interviews he always remained standing until asked to sit down. Though he had repeated many grades in school, he never presented a problem in discipline or truancy. Although the mother urged him to fight when necessary, he would not fight back. He was not a leader and was easily led by those whom he liked. He feared authority and usually submitted, i. e., to his father or teachers. He obeyed commands quicker than he responded to persuasion.

Evidence of Aggression.—He refused medication by hypodermic injection. He was facetious in interviews. In order to gain the family's attention, he talked in a loud voice about things, e. g., football scores, in which the others were not interested. He danced the Charleston in front of guests.

Characterization.—He was characterized as submissive.

CASE 14.—A boy aged 13 years and 3 months, the second of three children, who was in grade 7 A at school (a special class for reading disability) and had an intelligence quotient of 103, was referred because of poor school work and unpopularity with children.

Physical Findings.—The height was 56.8 inches (144 cm.) and the weight 101 pounds (45.8 Kg.) (24 per cent above the median). There was feminine distribution of fat. The fingers were tapering; the skin was smooth and velvety. There was slight pubic and no axillary hair. The penis was very small. The developmental history was normal.

Laboratory Tests.—The blood pressure was 106 systolic and 78 diastolic. The metabolic rate was —5 per cent.

Treatment.—Glandular treatment was given from the age of 8 to 10 years.

Evidence of Submission.—The mother said: "He is a coward. He sees that the doors are all locked at night, and he will not fight his battles with any one." Though she was nagging and exacting, he yielded fairly well to her discipline. He went to bed on time. Though he had much difficulty in school, he was never truant.

Evidence of Aggression.—He played chiefly with younger children, whom he dominated and tried to boss. He bullied the older sister. He was disobedient to his mother, chiefly in response to her tutoring and insistence that he read. Much aggression in the form of disobedience developed out of the difficulty in school. Several teachers said he was a pest and was not content "unless he had the center of the stage." Since coming to the institute he continually defied all authority and threatened his mother to tell the psychiatrist.

Characterization.—His behavior was characterized as mixed.

CASE 15.—A boy, aged 12 years and 6 months, the elder of two children, who was in grade 7 B at school and had an intelligence quotient of 125, was referred because he presented a disciplinary problem in school.

Physical Findings.—The height was 61.2 inches (155 cm.) and the weight 134 pounds (60.8 Kg.) (39 per cent above the median). There was feminine distribution of fat. The penis was short; the testes were normal. Genu valgum was present. The developmental history was normal, except that the patient was always restless and overactive and the testes were partially descended until the age of about 10 years.

Laboratory Tests.—The metabolic rate was —2 per cent.

Treatment.—At the age of 11 years pituitary and thyroid treatment was given and continued about six months.

Evidence of Submission.—There was none.

Evidence of Aggression.—The patient had always presented a disciplinary problem in school in spite of his high grades—talking, pushing boys in line and fighting. He was self-reliant and showed initiative in getting a job. He was disobedient at home. He had difficulty in making friends because he "always wanted to be the boss." He showed interest in girls. He was fond of sports. He talked freely in all interviews. (He was the only child in the family for nine and one-half years and had been spoiled.)

Characterization.—He was characterized as aggressive.

CASE 16.—A boy aged 14 years and 8 months, an only child, who was in grade 8 at school and had an intelligence quotient of 115, was referred because the school considered him a problem. He was lazy and "wandered around the halls."

Physical Findings.—The height was 66.2 inches (168 cm.) and the weight 190.2 pounds (86.3 Kg.) (52 per cent above the median). At the age of 10 years he weighed 200 pounds (90.7 Kg.). There was female distribution of fat. The testes were normal; the penis was somewhat undersized. The fingers were not tapering. The growth of hair was normal. According to the examiner, "the distribution of fat suggested hypopituitarism, but there was no genital infantilism or retardation in the developement of hair." The voice was high pitched. The developmental history was normal.

Evidence of Submission.—The patient showed lack of interest and energy. He was described as "well behaved, obedient and docile." He was helpful with the housework and was obedient at home. He took gibes of "Fatty" good-naturedly. He accepted his parent's directions readily.

Evidence of Aggression.—He was active in sports and played on the baseball team. He had several friends and was considered sociable.

Characterization.—He was characterized as submissive.

CASE 17.—A boy aged 14 years and 7 months, the eldest of three children, who was in grade 8 at school and had an intelligence quotient of 95, was referred because of retardation in school.

Physical Findings.—The height was 64 inches (162.5 cm.) and the weight 164 pounds (74.4 Kg.) (44 per cent above the median). At the age of 16 years and 1 month the height was 69 inches (175.3 cm.) and the weight 185.5 pounds (84.2 Kg.). There was female adiposity, with gynecomastia. The genitalia were normal. The developmental history was normal.

Laboratory Tests.—A roentgenogram showed that the sella turcica was enlarged. The metabolic rate was —6 per cent.

Evidence of Submission.—The father said that the patient was too soft and tender hearted and that when rebuked he cried. "He never gave any trouble at home or at school." He was always quiet and well behaved. The mother expressed the opinion that he was too much of a home boy. He cried when his brother or sister was scolded. He made no friends outside the home. The mother spoke frequently of his softness and babyishness. She had tried to coax him to play baseball with the boys, but he "refused to make a fool of himself by trying to bat a ball that he always missed." He avoided all sports. He was distinctly a "mother's boy." He was immature and obedient. At the time of the last contact with the institute he was doing well as a plasterer. (The mother was dominating.)

Evidence of Aggression.—There was none.

Characterization.—He was characterized as submissive.

CASE 18.—A boy aged 10 years and 2 months, the third of five children, who was in grade 7 B at school and had an intelligence quotient of 161, was referred for school placement by the visiting teacher because he was frequently late and untidy.

Physical Findings.—The height was 51.8 inches (131.5 cm.) and the weight 85 pounds (38.6 Kg.) (29 per cent above the median). The skin was soft and velvety. There was female distribution of fat, with gynecomastia. The fingers were tapering. Genu valgum was present. The penis was small. The appearance was feminine. The developmental history was normal, though the patient had always been "delicate."

Evidence of Submission.—He cried easily. He had made no friends. He had no interest in sports; he was absorbed in books and had no social initiative. He was submissive to the mother's corporal punishment and was obedient. The report from the camp (at the age of 10 years and 6 months) stated: "A follower . . . obedient, never antagonistic; friendly, not defiant; rather dreamy."

Evidence of Aggression.—He showed off his book knowledge and was at ease in interviews.

Characterization.—He was characterized as submissive.

CASE 19.—A boy aged 7 years and 5 months, the eldest of three children, who was in grade 1 B at school and had an intelligence quotient of 81, was referred because he was "ashamed," afraid to go to school and nervous.

Physical Findings.—The height was 51 inches (129.5 cm.) and the weight 74 pounds (33.6 Kg.) (40 per cent above the median). There was girdle adiposity. The genitals were normal. The fingers were tapering. The developmental history showed that the patient talked at 2½ years.

Laboratory Tests.—The metabolic rate was —3 per cent. The sella turcica was small, measuring 0.6 by 0.4 cm.

Evidence of Submission.—The patient was bashful, timid and secretive. He was afraid to go to school. He cried frequently, was afraid of strangers and did not stand up for himself with other children. The father said he was too submissive and had urged him to fight back when struck by other boys. The mother had visited the school to get the teacher to prevent other boys from striking him. He played chiefly with the younger sister and with two smaller boys. He was ordinarily submissive and gave up his toys readily to his little sister. He was always "a very good boy." When observed with his sister, aged 5 years, she seized things out of his hand and pulled down things he had made. He made no objection at any time.

Evidence of Aggression.—There was none.

Characterization.—He was characterized as submissive.

CASE 20.—A boy aged 12 years and 6 months, the youngest of three children, who was in grade 7 A at school and had an intelligence quotient of 122, was referred because of fears.

Physical Findings.—The height was 59.5 inches (151 cm.) and the weight 108.5 pounds (49.2 Kg.) (17 per cent above the median). There was girdle adiposity. The skin was soft and smooth. The penis was short, and the testes were small (the size of a pea). The fingers were tapering.

Laboratory Tests.—A roentgenogram of the sella revealed normal structure (1.2 by 0.9 cm. on a 28 inch [71.12 cm.] film).

Evidence of Submission.—The patient's fears were of death, noises at night, disease and deformity, and going to sleep alone. He played only with smaller

children. He was afraid to fight, though he had many word battles. When angry at home he left the room and wept. He was submissive with teachers, "accepting their authority without an argument." At home he was obedient but battered down the parents' resistance with excuses and arguments. He definitely preferred girls to boys. He was overly sensitive and easily hurt and afraid to go to doctors and dentists. He spent most of his time playing with toys at home. When forced to go out to play, he chose younger boys. At camp, at the age of 9 years, he threatened suicide if he was not sent home. In school he was "grouped naturally with the younger children." He "allowed himself to be dominated by a girl cousin 1 year older than he; he accepted her judgment and was unhappy for hours when she rebuffed him." In spite of teasing, he was happiest when playing alone with tin soldiers, boats, trains and games of younger children. When he escaped detection, he remained in his bath for hours, floating tops, birds, soap and clothespins on the water. He yielded to infantilizing care; e. g., he did not dress himself until the age of 12 years.

Evidence of Aggression.—On the basis of his phobias, he stubbornly refused to take medicine or to sleep alone. He tried to dominate younger children. He was spontaneous and outspoken in interviews. He showed nagging insistence on being with his older sisters and was a pest in arguing to get what he wished.

Characterization.—He was characterized as submissive.

CASE 21.—A boy aged 12 years and 1 month, the eldest of three children, who was in grade 7 A at school and had an intelligence quotient of 130, was referred because of laziness and poor school work, in spite of his high intelligence.

Physical Findings.—The height was 60.7 inches (154 cm.) and the weight 133.5 pounds (60.55 Kg.) (38 per cent above the median). There was girdle adiposity. The skin was smooth; the fingers were tapering. The genitalia were normal. Pubic hair was present. "The distribution of fat suggested a hypopituitary condition." On physical examination at the age of 14 years and 4 months the height was 67.5 inches (171 cm.) and the weight 180 pounds (81.6 Kg.) (48 per cent above the median). Girdle adiposity was present. There was a moderate growth of pubic hair.

Laboratory Tests.—The metabolic rate was +18 per cent, and one month later, +1 per cent.

Evidence of Submission.—The patient was obedient to the father. He yielded to his parents' refusal to let him take risks, e. g., roller-skating, baseball, etc. A typical reply was: "But mother wouldn't allow it." In spite of disliking school, his conduct in class was good, and he was never truant. He was interested in sports but was prevented from being active because of the oversolicitude of the parents.

Evidence of Aggression.—He quarreled with the younger sister. He was disobedient to his mother, i. e., indifferent to her nagging. He had many friends and, though not a leader, made easy contacts. He was frank and outspoken in interviews.

Characterization.—He was characterized as submissive.

CASE 22.—A boy aged 13 years and 4 months, the younger of two children, who was in the first year of high school and had an intelligence quotient of 114, was referred because of his lack of friends and timidity.

Physical Findings.—The height was 58.5 inches (148.5 cm.) and the weight 103 pounds (46.7 Kg.) (15 per cent above the median). There were girdle obesity and beginning development of pubic hair. The voice was high pitched. The fingers were tapering.

Evidence of Submission.—The patient worked hard in school and displayed excellent conduct; i. e., he was a "plodder." He loved housework and did it voluntarily. He was shy and timid and never played on the street with other boys. In the mental testing room he was described as "soft spoken, compliant and docile." The boys called him "sissy," "picked on" him and took his money from him; he did not defend himself. He begged his mother to walk to school with him and still slept with her. He never had quarrels with his brother. He was obedient and affectionate to the parents. His only interests were reading and music. He yielded to the mother's infantilizing care, even going to bed as a punishment in the afternoon. He showed a remarkable lack of initiative, enthusiasm or aggression.

Evidence of Aggression.—None was presented.

Characterization.—He was characterized as submissive.

CASE 23.—A boy aged 10 years and 4 months, the second of three children, who was in grade 3 B at school and had an intelligence quotient of 96, was referred because of retardation in school and slowness.

Physical Findings.—The height was 56 inches (142 cm.) and the weight 103 pounds (46.7 Kg.) (32 per cent above the median). There was girdle obesity, with gynecomastia. The skin was smooth and moist. The penis was small (length 1 inch [2.54 cm.]); the testes were normal.

Evidence of Submission.—The boy had no friends and kept away from other children. Though he was in a special class for retarded children and had repeated grades in school, he showed good conduct in school and was never truant. With adults he was obedient, shy and babyish. He was easily frightened. He was obedient with the parents and allowed the mother to dress him. (The general picture was that of a submissive, feebleminded boy, in spite of the intelligence quotient of 96, with fanciful boasting.)

Evidence of Aggression.—He fought with younger siblings and boasted about his fighting ability.

Characterization.—He was characterized as submissive.

CASE 24.—A boy aged 10 years and 4 months, the elder of two children, who was in grade 5 A at school and had an intelligence quotient of 115, was referred because he had momentary spells in which he became flushed and lost his breath (later, attacks of petit and grand mal) and enuresis.

Physical Findings.—The height was 57.2 inches (145 cm.) and the weight 87 pounds (39.5 Kg.) (10 per cent above the median). There was girdle adiposity, with gynecomastia. The fingers were tapering, and the skin was smooth and velvety. The penis was very small; the testes were normal.

Evidence of Submission.—He did not play with boys, for the mother would not allow it. He yielded to her numerous demands as to when, where and how he should play and to her helping him dress, etc. He was obedient to his parents. He showed good conduct in school. He had no interest in sports and was called a "sissy." (The general picture was that of a boy submissive to a dominating mother.)

Evidence of Aggression.—There was one episode of stealing and one instance of truancy.

Characterization.—He was characterized as submissive.

CASE 25.—A boy aged 9 years and 9 months, the eldest of four children, who was in grade 4 A in school and had an intelligence quotient of 101, was referred

because of rebelliousness at home. He was disobedient to and struck his mother and fought with the siblings.

Physical Findings.—The height was 54.5 inches (138 cm.) and the weight 97 pounds (44 Kg.) (37 per cent above the median). There was girdle obesity. The penis was small (less than 1 inch [2.54 cm.]) in length; the testes were small (size of a pea). The fingers were tapering. The patient showed frank and marked sensitivity to the smallness of the genitals.

Evidence of Submission.—He was a "model boy" in school.

Evidence of Aggression.—He hit his mother and fought with the brother and other children. He assumed a dominating position in the home. There had been "uncontrollable temper tantrums" at home since the age of 3 years. He bullied other children, stole and was truant. (He was the oldest boy and was overprotected in infancy. The father was out of the home.)

Characterization.—He was characterized as aggressive.

CASE 26.—A boy aged 9 years and 7 months, an only child, who was in grade 4 A at school and had an intelligence quotient of 97, was referred because of enuresis and stealing.

Physical Findings.—The height was 58.2 inches (147.8 cm.) and the weight 104 pounds (47.2 Kg.) (24 per cent above the median). There was girdle adiposity. The penis was small, and only one testicle was descended. On examination at the age of 10 years and 2 months, the height was 59.3 inches (150.6 cm.) and the weight 115 pounds (52.1 Kg.); the testes were both in the scrotum.

Laboratory Tests.—A roentgenogram of the sella revealed normal structure. The metabolic rate was +17 per cent.

Evidence of Submission.—He yielded good-naturedly to the nickname of "fat horse." When teased and hit by other children he would not fight back unless the assailant was smaller than himself. He was afraid to fight, though urged to do so by the parents. He played with children younger than himself; children of his own age would not play with him because he was "awkward." He yielded to the mother's infantilizing care, i. e., bathing and dressing, and also to her exacting discipline, e. g., rewriting lessons, practicing the violin, returning home fifteen minutes after school or playing only in front of the house.

Evidence of Aggression.—He was disobedient to his parents (?). He stole and bullied younger children. He talked freely with strangers and was boastful in interviews.

Characterization.—He was characterized as submissive.

CASE 27.—A boy aged 11 years and 5 months, an only child, who was in grade 4 B at school and had an intelligence quotient of 83, was referred because of retardation in school.

Physical Findings.—The height was 55.8 inches (141.6 cm.) and the weight 89.5 pounds (40.6 Kg.) (15 per cent above the median). There was girdle adiposity, with gynecomastia. The skin was smooth and moist. The genitalia were "rather small." He had been fat since the age of 2 years.

Evidence of Submission.—Though he disliked school and had to repeat several grades, there was no truancy, and his conduct was A. When teased by other children he did not answer back or fight. He was obedient to his parents. He disliked sports. He was criticized for not being aggressive with friends and for not standing up for his rights. He had to be pushed and encouraged to do things.

Evidence of Aggression.—He had a number of friends. He liked baseball.

Characterization.—He was characterized as submissive.

CASE 28.—A boy aged 9 years and 4 months, the elder of two children, who was in grade 4 A at school and had an intelligence quotient of 105, was referred because of stealing and effeminacy.

Physical Findings.—The height was 54.5 inches (138 cm.) and the weight 78.5 pounds (35.6 Kg.) (10 per cent above the median). There was girdle obesity, with gynecomastia. The skin was soft and smooth. The penis was small, and the testes were normal. The fingers were tapering. The mannerisms were effeminate. There had been developmental delay in walking (at 19 months) and talking (at 24 months).

Evidence of Submission.—The patient did not play any boys' games. He was obedient to his parents. He never played with boys and did not want to; he preferred playing with girls. His conduct in school was excellent. He enjoyed helping the mother with the housework, especially washing dishes and dusting. In recent months, when the mother said she did not love him (as punishment for criticizing the baby), he sobbed most of the day. He was afraid to fight, and the younger brother, aged 5 years, defended him from the boys, saying he could not defend himself. As a baby he showed general lack of energy and was "too lazy even to cry." He was slow, lazy and forgetful.

Evidence of Aggression.—He fought with the younger brother and stole money from his parents.

Characterization.—He was characterized as submissive.

CASE 29.—A boy aged 15 years and 5 months, the youngest of three children, who was in the first year of high school and had an intelligence quotient of 111, was referred because of poor school work and laziness.

Physical Findings.—The height was 62.2 inches (158 cm.) and the weight 168 pounds (76.2 Kg.) (32 per cent above median). There was girdle obesity. The skin was smooth. There was a small amount of pubic hair. The genitals were small. The mannerisms were effeminate. Onset of the obesity occurred at the age of 13 years.

Laboratory Tests.—The metabolic rate was —16 per cent.

Evidence of Submission.—He was "teacher's pet in all the grades." He was obedient to the mother. Though he strongly disliked school and failed in all subjects, he was never a truant. He did not fight with siblings. He had numerous fears, such as of danger and disease. He was always "polite and mannerly."

Evidence of Aggression.—He was sociable and had many friends until about the age of 14, since which time he dropped them and spent all his leisure at home reading and talking to his mother. He liked swimming. (The difficulties had their onset at about the age of 13 to 14 years.)

Characterization.—He was characterized as submissive.

CASE 30.—A boy aged 8 years and 7 months, the elder of two children, who was in grade 4 B at school and had an intelligence quotient of 122, was referred because of soiling, laziness and untidiness.

Physical Findings.—The height was 52.5 inches (133 cm.) and the weight 86 pounds (39 Kg.) (28 per cent above the median). He was quiet, obese and slow moving. The adiposity was increased over the breasts, thighs and buttocks. The fingers were tapering. The genitalia were normal, and there was no developmental delay except in teething.

Evidence of Submission.—In school he did not play with others, even when the teacher urged him. At home he played with other boys but had to be encouraged to "mix in." In the first interview he sat mostly on the edge of the chair and appeared shy and reticent, though later he became spontaneous in con-

versation. He was slow and quiet. He complained that he did not care to play with the boys at school because they were too rough. He was shy and sensitive and cried a great deal. He cried when his mother scolded him or when he was made to stop playing. He was described as "a quiet, pleasant and obedient child, who went to bed immediately and without protest when it was time." He was obedient to both parents. When the mother was ill the patient was quiet and tiptoed about. He played nicely with the younger sister and seldom quarreled with her.

Evidence of Aggression.—None was present.

Characterization.—He was characterized as submissive.

CASE 31.—A boy aged 11 years and 2 months, the younger of two children, with an intelligence quotient of 90, was referred because of retardation in school.

Physical Findings.—The height was 55.3 inches (140 cm.) and the weight 101.5 pounds (46.05 Kg.) (38 per cent above the median). There was increased adiposity over the breasts, lower portion of the abdomen and buttocks. The fingers did not taper. The genitalia were "relatively small."

Laboratory Tests.—The metabolic rate was normal. A roentgenogram showed the sella to be small.

Evidence of Submission.—The conduct in school was good, and there was no problem in discipline or truancy, in spite of the retardation.

Evidence of Aggression.—The patient was quarrelsome and fought with his sister. He was disobedient to the mother. There was one instance of his running away from home. He belonged to a gang of delinquent boys and was active in baseball, etc.

Characterization.—He was characterized as aggressive.

CASE 32.—A boy aged 12 years and 10 months, the elder of two children, who was in grade 7 A at school and had an intelligence quotient of 112, was referred because he did poor work in school, preferred younger children and was unpopular.

Physical Findings.—The height was 64 inches (162.5 cm.) and the weight 115.5 pounds (52.35 Kg.) (5 per cent above the median). There was girdle obesity, the distribution of fat being of the female type. The penis was small, and the testes were normal. The fingers were long and tapering. Glandular study was recommended.

Evidence of Submission.—The patient was unpopular and afraid to join in games. He preferred younger children. He cried easily. He was slow in school and was unhappy there, but there was no truancy or tardiness.

Evidence of Aggression.—None was presented.

Characterization.—He was characterized as submissive.

CASE 33.—A boy aged 10 years and 4 months, the elder of two children, who was in grade 4 B at school and had an intelligence quotient of 69, was referred because of failure in school, dependence and fear of other children.

Physical Findings.—The height was 55.5 inches (135.8 cm.) and the weight 75 pounds (34 Kg.) (average). There was some increase of adiposity about the breasts, hips and abdomen. The penis was very small, and the scrotum was also small.

Evidence of Submission.—He was obedient as a rule. He was sensitive and cried easily. He gave no trouble in school, except that his behavior was sometimes infantile. He feared other children.

Evidence of Aggression.—None was presented.

Characterization.—He was characterized as submissive.

AMPHITHYMIA

SOME SYNDROMES OF DEPRESSION AND ELATION

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The manic-depressive psychosis is not a new subject for psychiatric observation or psychoanalytic formulation, nor is the cyclothymic personality without some excellent discussion. Manic-depressive psychosis is defined as a severe chronic mental disorder characterized by strikingly dissimilar phases which may follow each other with or without intervals of normality or may recur without the contrasting phase; the one phase includes acceleration of thought, hyperactivity, flight of ideas and distractibility, with resulting serious disturbances of judgment; the other phase is defined by poverty of ideation, hypo-activity and restriction of thought to a few grief-provoking ideas. The cyclothymic personality is viewed generally as an incomplete version of the psychosis, an abortive form of it or the manifestation of its anlage.

The clinical material of the Cook County Psychopathic Hospital that has come to my attention during the past fifteen years has included many cases in which the condition was diagnosed according to these rubrics, a considerable number of which were readily demonstrated to be instances of paranoid schizophrenia or excitement in the course of catatonic illness. A glimpse below the surface of the superficial symptomatology usually sufficed for this discrimination. Content and processes foreign to the manic-depressive picture came readily to life. In this connection the psychoanalytically oriented psychiatrist is less inclined to postulate "mixed states," for he considers the personality system and its current adaptation to the personal environment in terms of genetic evolution and more distant goals rather than merely as patterns of overt behavior. The way in which the patient experiences his personal reality and the reality of others is interpreted in the light of the needs or necessities that it serves within the configuration of the patient and his environment, as this configuration has come to be. With these considerations in mind, one can generally establish the diagnosis of accessible patients with psychosis without much uncertainty.

Occasionally in the hospital and more frequently in private consultation, one sees excited or depressed patients whose condition seems, from experience in their psychoanalysis, to be but little related either to manic-depressive psychosis or to schizophrenia. The condition of most of these patients falls under the psychiatric rubric of the psycho-

pathic personality; they are persons who at no time in life have had a good grasp of reality, either their own or that of others. They seem to have many attributes of culture in its literary sense; they know the right answers to the usual situations and are often rather genial persons, but they have almost no ability to profit from experience. This tendency to act on impulses without consideration for realities, coupled with swings of mood, has led exasperated relatives or friends, during the period of greatest hyperactivity or of most voluble depression, to bring them for treatment. In private practice I have also come into contact with persons whose disturbances have been given a manic-depressive label and seem related to the type just mentioned but whose ability to learn from experience is much more marked. There are persons who during the greater part of their lives have been efficient professionally and socially but who have also been subject to breakdown. It has occurred to me that the gross symptom of swings of mood—of phases of hyperactivity and depression—common to all these patients, a symptom which has actually given title to a group of psychotic phenomena, has made it all too easy to assume that all these personalities represent some degree of development of a manic-depressive phenomenon. Inquiry into the exact nature of the moods has constantly brought forth a group of factors which are fundamentally different from those in the classic manic-depressive psychosis. I believe that an adequate differentiation of this group will help to clarify understanding of psychotic disturbances and of the cyclothymic personality. I know from experience with a number of patients with a condition of this type that the discrimination is of real importance in formulating one's therapeutic approaches and expectancies.

It is well known that different personalities may make use of one and the same group of processes to subserve widely different aims. I wish here to emphasize that the syndrome of more or less cyclic or otherwise recurrent periods of excitement and depression in some persons manifests processes and configurations of personality quite unlike those of the better known clinical pictures. I shall refer to these as the amphithymic personality, or the amphithymic character, to indicate that their outstanding pathologic characteristic is a swing of mood, without any particular relationship to the manic-depressive and related cyclothymic pictures.

It is to be understood that all phases of depression, whether in manic-depressive insanity, schizophrenia or psychoneurosis or in the usual situations of grief, are objectively similar, as are also all incidents of elation. In depression there are slowing of all activity, loss of interest in one's business or profession, reluctance to make social contact, loss of self-esteem and some feeling of hopelessness—of "what's the use of all this?" These may be manifested by silence or self-recrimination,

by physical lethargy or wringing of hands, by tears or wailing or by suggestions that the subject might as well "end it all" or that he wishes some one else would "put him out of his misery." These manifestations are subject to almost as many variations of elaboration or restraint as there are persons suffering them.

However, in true manic-depressive psychosis one sees depression in the extreme—silence and lethargy may sometimes progress to an almost complete inhibition of activity. Expressions of grief, weeping and the other manifestations may assume the most aggravated and exhausting character, requiring sedatives to prevent the patient from serious physical damage to himself. Sense of inferiority and self-blame take on the fervor of religious penance for sins the patient feels can never be forgiven. Hopelessness is such that there is little or no cooperation with efforts toward assistance and relief. "Ending it all" is often no figure of speech but the only solution to a hopeless problem and, more important, not infrequently the price the patient feels he has to pay for the horrible sins he believes he has committed. Only by self-destruction can he be absolved. These patients are the "picture of grief and misery;" their loss of appetite has resulted in serious decrease in weight; they often suffer insomnia and constipation. Their thoughts are few; their activity is at a minimum; their tendency to self-destruction is great, and the depression usually lasts over a long period.

The general psychoanalytic theory [1] sees in the development of the manic-depressive psychosis a conflict between a guilty ego and a severe superego—starting with an early severe narcissistic blow, the patient has become fixated on an oral aggressive level, suffering from severe ambivalence toward his objects; any situation which increases his feeling of inability to love and so to be loved also increases his hostility toward his objects, and the hated object is introjected on to the ego and is, as it were, attacked by a severely criticizing superego. The self-destructiveness of these patients is explained, therefore, as the need for hospitality toward the invaded ego. Attention may now be turned to the patterns to be observed in the depression of amphithymia.

DIFFERENTIATION IN THE PHASE OF DEPRESSION

Inquiry into the nature of the depression of patients with amphithymia reveals several interesting contrasts to the true depressions just described. First, the patients do not look depressed; they appear only sullen and hostile. They resemble petulant children, angry at a world

1. (*a*) Freud, Sigmund: Mourning and Melancholia, in Collected Papers, translated by J. Rivière, London, Hogarth Press, 1925, vol. 4. (*b*) Abraham, Karl: Zentralbl. f. Psychoanal. **2**:302, 1911; Internat. Ztschr. f. Psychoanal. **4**:71, 1916. (*c*) Radó, Sandor: The Problem of Melancholia, ibid. **9**:369, 1923.

which has not gratified their demands. Second, although they complain of loss of appetite and difficulties with sleep they look well nourished and little fatigued. During treatment I have found that sleep is seldom materially disturbed. By changing hours for sleep, going to sleep at a later hour and napping during the day, these patients contrive to simulate insomnia after a fashion. One patient frequently slept for from fifteen or twenty minutes during the hour for analysis. None of the patients lost a great amount of weight, and two gained during the depressions. None suffered from constipation, but some had diarrhea. The patients show little if any diminution of sexual drive during the depressions. Several continued heterosexual activity. One patient in the midst of a so-called depression returned from a week-end almost in triumph, with the information that he had had intercourse with a virgin. A great deal of masturbatory activity is characteristic. Crying rarely occurs in these depressions; when it does, it is usually associated with anger at the frustration of a demand.

The history of the disorder also arouses suspicion, for the frequency of the attacks is not characteristic of true manic-depressive psychosis. One patient came to analysis after having had six attacks of depression and six of elation in three years. During the first year of analysis he had two so-called periods of depression and three of excitement. Two patients had had a period of depression of several months' duration each year for two and three years, respectively. While this frequency is not found in all cases, the probability is that when it is present the condition does not fit the classic manic-depressive picture. Associated with the abnormal frequency of attack is the extraordinary fact that an attack can be aborted occasionally by methods that do not work in cases of manic-depressive psychosis. One patient in the depths of his depression recovered almost completely when relieved of all responsibility and sent to a camp where he was taken care of and waited on. Another who showed signs of going into a depression was deterred by the remark: "Do you plan to have this a ten day or a three month depression?"

The self-accusations of the patient with amphithymia have rather a hollow sound. They are closely related to factors in the patient's actual situation; they are appropriate and rarely extravagant. One hears expressions of remorse over neglect of duties or over sprees—social, sexual or financial—which have occurred during the preceding hyperactive phase and regrets for hostilities that have been acted out. The remorse and regrets take forms like these: "I won't be able to go back to my practice because I don't really know enough to practice honestly." "I will never again be accepted socially because I've made a spectacle of myself." "I'll have to move to another town because I feel sure my clandestine sexual affairs have been discovered."

Not only are the expressions of remorse, regret, self-accusation and criticism mild exaggerations of factual situations, but one often hears with them evidences of concomitant fantasies of omnipotence and insistence on special potentialities which should exempt the patient from ordinary demands. He does not need to study or work as ordinary persons do to achieve his aims; he believes that he is intended for big things and great honor. One patient, a physician, insisted that he was destined to be a great psychiatrist. When it was pointed out that, aside from the experience of his own instability, he had no training in that field, he replied that his special ability made him capable of success in psychiatric practice without the opportunities for training which his analyst had had. The hopelessness of these patients often results not because the patient feels any real lack in himself but because all psychiatrists—especially his own—are charlatans and because the methods employed, though perhaps useful to others, are inadequate to his unusual needs. The patient is not hopeless; the physician is. Blame for the immediate disorder is often not self-blame but accusations against the environment. One patient felt that the boredom of his home and his parents' constant bickering were responsible for his depression. Another declared that her family's criticism drove her to depression. A third stated that her father's unjust accusations and punishments were the cause of her depression.

The environment is often found wanting, because persons of this character all make unreasonable demands on it—another feature well demonstrated during the depressions. Unlike true manic-depressive patients, who feel they are beyond aid or do not deserve it, these patients demand a fantastic amount of personal attention from those about them. To be waited on hand and foot is a literal request. They want to be told how fine they are, how capable, how lovable; they want each moment of the day planned; their constant plea in treatment is: "You must help me," "you must tell me what to do." They demand financial support—from insurance companies or parents—and are constantly worried whether or not it will be forthcoming. One patient with an income of his own constantly railed about his father's stinginess, although the father was paying all his bills. Another, who was living on his accident and health insurance, regularly put on an act of profound dejection for the benefit of the insurance investigator, although he too had sufficient reserves to pay his own bills.

This demand for care is inextricably linked with a demand for preferment, for being the exception. The patient with amphithymia wants not only care and affection but more care and affection than any one else. He wants not only to be told what to do but to have the way paved for his accomplishment, without any effort or cost to himself. He is extremely jealous of attention or recognition of any one

about him; for treatment he wants an hour exactly coinciding with his own convenience. In several instances, patients have made great effort to contrive to get treatment free of charge. One lost his job through unconscious intent to avoid paying for analysis. He formulated two reasons: He would be getting something for nothing, and he believed that he would be the only patient being treated under those conditions. Another regularly forgot to pay his bills; a third patient's insistence on my fraudulence was motivated by the thought that under those circumstances I could not collect from him or his father, who was paying my bill. To get something for nothing and to be the only one receiving care without cost are characteristic of these persons. It is difficult to give credence to the sincerity of their self-criticism and their insistence that they are of no value and never will be, when the least encouragement brings out the facts just described.

An important distinction between these patients and those with true manic-depressive psychosis is the degree of awareness of the hostilities and of the gratification of these hostilities in the depressions. I have already pointed out that the self-blame is superficial and that the accusations against the environment are numerous and their demands excessive. Their accusations range from hypercriticism of every one about them to virulent vituperation. I have been called almost everything unpleasant under the sun by these persons during the depressions. In several instances the patient was aware that his silence and lack of sociability were merely inhibitions of his hostility—if he spoke at all his words would be unacceptable—the inhibition resulted from fear of retaliation by the object of his aggression. Dreams are often clearly destructive. The depression and helplessness of these persons are themselves constantly used for aggressive purposes. By being helpless one can demand attention; one can interrupt the lives of others; one can deprive people of money, pleasure and peace; one can arouse fears and make them concerned for one. Suicidal threats and attempts are almost always gestures to achieve this end and are often used to force the parent or wife or psychiatrist to send the patient to an institution.

This wareness of hostility on the part of patients with amphithymia and the at least partial realization that the activity is calculated to gratify aggressive aims, covered by protestations of helplessness, misery, inferiority and the accouterments of depression, result in an impression of fraudulence so marked that I have from time to time referred to patients of this type as "fraudulent characters." They show unquestionable appreciation of the secondary gains from the depressed state. Passive aggression is extremely effective but easy to deny. Their nuisance value brings the environment to terms. However, the picture is not quite as clearcut as I have perhaps suggested. It is often difficult to tell how much of the purposiveness is wilful activity and is clearly

perceived consciously. Sometimes the patients give the impression that activity deliberately initiated has passed out of control and that, although there is some awareness of what they are doing, they are really unable to check the behavior. In connection with this state of affairs I once coined the phrase "unconscious malingering." One finds not infrequently that there is neither straightforward purposive activity nor furtive behavior calculated to satisfy the patient's needs and desires, nor yet behavior the implications of which the patient is completely unaware. These patterns are blended, and from this comes the impression of fraudulence which distinguishes these patients from those with real manic-depressive psychosis.

Another important characteristic of patients with amphithymia which distinguishes them rather sharply from true manic-depressive patients in depression is the marked "will to live." They are not essentially self-destructive; they have every wish to continue existence. The depression seems directed not to end a hopeless and guilty life but to achieve an existence freed from responsibility and unpleasant duties. The depression seems largely to be the source of satisfaction in being taken care of, to arise from the desire for dependence and to manifest a determination to have some one minister to the patient's needs. Institutional care is welcomed if more personal nursing is not available. In patients with true depression one feels that the mood is primarily a matter of self-torture, with incidental gratification. The depression of the amphithymic person is essentially gratification, with minimal self-torture. This is of real clinical importance. Obviously, if gratification rather than punishment is the essential issue in such depressions there is much less danger of suicide. In my opinion, the risk of self-destruction for the person with amphithymia is minimal and does not ordinarily contraindicate treatment outside an institution. In these cases suicide is often used to frighten the psychiatrist. An order for the supervision of the patient or his institutionalization may seriously jeopardize any possibility of subsequent therapeutic change when it can be interpreted by the patient as a success of such threats. Other factors in these cases should be used to determine the extent of the risk. The urge to live, I repeat, is strong; what the patient wants are gratuities of all kinds, money, service and protection; in short, he is depressed because he has not been able to achieve a life of passive gratification and resents the world that refuses him.

DIFFERENTIATION IN THE MANIC PHASE

Manic attacks have been described as periods of hyperactivity, acceleration of thought, with accompanying flight of ideas and distractibility, serious disturbances of judgment, release from inhibition and exalted

ideas of self—periods of jubilee and self-congratulation—a triumph of ego over superego and a celebration of the event. The so-called mania of persons with amphithymia shares with all forms of excitement the characteristics of hyperactivity, acceleration of thought, appearance of cheer and good spirits, a degree of release from inhibition and a flattering opinion of self. But in the mania, as in the depression, these persons indulge in behavior strikingly different from that of the patient with true manic-depressive psychosis.

Among other variations one finds on investigation that the person with amphithymia is not essentially cerebrating a victory over superego or the introjected hated object or anything else. There is no real jubilee but, again, as in the depressions, a gratification of the oral-anal sadistic tendencies in threats, malicious lying and aggressive sexual acts and, occasionally, in physical violence. The patients are hyperactive, but one may note that they suffer no sustained mania. They are capable of highly purposive activity and frequently carry on their work, or more frequently their social life, without arousing comment among friends. They are talkative, and one sometimes feels that their talk is compulsive, but there is no flight of ideas. Serious disturbances in judgment are not characteristic. Often the so-called manic acts occur only after frustration of demands, exactly as one may see temper tantrums follow refusal to a demanding child. Like the behavior during the depressions, these persons demand excessive attention in the excitement, and just as they use the helplessness of depression to gratify their demands, they use the hyperactivity of the so-called mania. One patient prepared the way for a manic outburst and for preferment during the attack by making friends with the staff in the hospital, which would receive him in such an event, giving them liquor and cigarets. When he entered the institution he was but mildly disturbed; he became unmanageable only when denied special foods, quarters and privileges. Two others exactly repeated this pattern, becoming violent only after a week or so of testing those about them and of dispensing bribes, at the end of which time they found themselves no further toward special privileges than before.

The indulgence of hostilities is no less conscious in this phase than in the depression; in fact, the mania consists primarily of a flagrant acting out of these hostilities. One patient said that she let her hostilities pile up during a depressed phase till she could not stand it any longer and then began to act them out directly in a phase of hyperactivity. Aggressive sexual acts are deliberate and are often carefully concealed, owing to conscious awareness of the personal dangers involving the patient's reputation. The aforementioned patient openly pursued the men around the hospital and was exhibitionistic with the psychiatrist. Another patient frequently took men out and made them drunk, while

pretending to become so himself, as a preliminary to masturbating them. Drink was served to lower resistance to the act and to render the men incapable of a counterattack. Another warned a friend that if he should ever become excited and want to fight the friend should accede to the demand but should temper his blows. A half-hour later the patient challenged the friend, who kept faith with him, while the patient hauled off with all the strength he could muster and beat the friend badly. All these persons indulge during the manic phase in malicious lying and threats which take on the color of blackmail. They publicly attack the reputation of family, friends and, especially, psychiatrists. One patient constantly threatened to ruin his father by telling the government facts affecting the father's income tax return. Another patient persuaded a nurse to give him special attention and later reported her as having tried to seduce him, for which she was dismissed.

Indications that these persons are not really celebrating a triumph of their omnipotence lie in the fact that much of the hyperactivity is designed not to demonstrate superiority and power but to convince themselves and others that they possess these qualities. Many spend money extravagantly but not with the cheerful abandon of one possessed of millions and eager to toss them away. They are aware of every cent they spend; they are consistently niggardly about money where others are concerned and spend only on themselves and where it will make a good public impression. They often plunge themselves into a whirl of social activities—large and elaborate parties calculated to get their pictures in the papers. They pursue celebrities to convince themselves that they are important persons around whom famous people gather. Much of the lying is directed toward building up a saga of their own importance. Not infrequently these episodes of hyperactivity are associated with excessive drinking and other methods of artificially inducing or maintaining a feeling of exhilaration. This need for reenforcement of the sense of well-being leads one to suggest that these patients lack the feeling of confidence and security characteristic of true manic attacks. Indeed, there is a more important relationship than the mere consumption of liquor between these persons and certain persons addicted to the use of alcohol. It has already been stated that several patients not only prepared the way for privileges in future attacks by establishing a connection with institutions during normal periods but in many instances were eager for institutionalization. Several institutionalized themselves. It is interesting, too, that the hyperactivity outside the institution is rarely violent. They show excitement, enough to wear down the persons around them and to induce their being sent to a sanatorium; it is only after admission to the institution that any really violent behavior occurs. One gets the impression from these patients that the mania is a cover to permit aggressive acts, the thought being somewhat as

follows: "If I am sufficiently manic to be placed in an institution I cannot justly be held responsible for my behavior." When incorrigible behavior coupled with excessive spending of money finally drove one patient's family to arrange for care at a sanatorium, the patient said: "They think I don't know what I'm doing. Why, I always know when I've overdrawn my account. I don't even bother to list the checks in my check-book but take them out of the back of the book. They are sending me away because they think I'm crazy. All right. I'll be crazy; I'll rave and smash furniture and have a grand time."

Thus, it seems that these patients resort to a manic phase exactly as some persons addicted to the use of alcohol resort to a "binge," in order to have the excuse of unawareness in drunkenness for the carrying out of forbidden activity. An alcoholic person of this type often alternates sprees with depression, the depression being a period of remorse and anxiety concerning the consequences of the asocial behavior. I have a patient under analysis who came to me for treatment for alcoholism. With treatment he revealed many characteristics of the person with amphithymia—among them, three periods of depression. There is good reason to believe that the alcoholic sprees corresponded with the period of elation of a person with amphithymia. A second patient whom I supervised entered a sanatorium with a diagnosis of manic-depressive psychosis. He entered during a period of depression, which lasted several months and was followed by a period of alcoholism. He, too, revealed mechanisms with treatment similar to those of the patients described in this paper. It seems that patients with amphithymia and certain persons with alcoholism are closely related and reveal similar mechanisms;[2] it is probable that this amounts to a clinical identity.

All the activity just discussed appears as purposeful and as carried out with complete awareness much of the time. Again, as from the behavior in depression, one sometimes obtains the impression that the hyperactivity has really passed out of control or that occasionally the purpose behind the act is just out of consciousness, for in these instances it is often perceived immediately after the act. Both in the mania and in the depression of the patient with amphithymia one can observe something of a fraudulent quality, which distinguishes them sharply from the true manic-depressive types.

SUMMARY OF DIFFERENTIATION OF AMPHITHYMIC TYPES OF DEPRESSION AND MANIA

It may be well, perhaps, to list the manifestations of depression and mania that distinguish the personalities of the type I have called amphithymic from those of the true manic-depressive type. In depres-

2. Radó.[1c]

sion the patient presents a petulant and hostile rather than a melancholy expression. In general, he suffers none of the vegetative disturbances, insomnia, anorexia, loss of weight, loss of potency and constipation.

The frequency of the attacks is suggestive, as is the occasional success in aborting such attacks by fairly simple means. Self-accusation is practically indistinguishable from anxiety over retribution for asocial activity and is associated with expressions of overvaluation of self. Hostility toward objects is consciously expressed and gratified. These persons, even in depression, make excessive demands for care, protection, attention and preferment. There is great awareness of the secondary gains from the depression. They show a marked will to live, and tendencies to self-destruction are minimal.

In the so-called manic phase the patients are hyperactive but do not maintain a manic state. They are capable of purposive activity, suffer no great loss of judgment and have no flight of ideas. The behavior is flagrantly, and frequently consciously, aggressive to objects. There is no real elation, and not infrequently they resort to alcohol to initiate or sustain a feeling of exhilaration. There is a release of inhibition, but they are not really free from "conscience activity." In both phases there are purposiveness and awareness not characteristic of the true manic-depressive type, which leaves one with the strong impression that the behavior is a simulation of depression or mania calculated to cover the gratification of oral and anal aggressive needs and excessive passive demands.

GENETIC CONSIDERATIONS

The background and genetic development of patients with amphithymia were surprisingly similar in all the cases that I studied.

The material of this study consisted of twelve patients who underwent analysis, eight of my own and four whom I followed as a supervising psychoanalyst. In each case a diagnosis of manic-depressive psychosis had been made prior to treatment, in several instances by a psychoanalytically oriented physician. I mention this to emphasize the significance of the differentiation of amphithymia: All these patients sought aid in the phase of depression; none made a serious attempt at suicide in the whole course of the analysis.

Analysis revealed: 1. The parent of the other sex was domineering, ultraconservative or sadistic, without consistently demonstrated affection, while the parent of the same sex was in some sense weak and failed to offer any neutralizing love to the child.

2. The family configuration showed no consistent demonstration of affection of parent for parent and often included frank sadomasochistic relations.

3. A specious religiosity or pretentiousness occurred with some frequency, usually in the parent of the other sex, with passive cooperation by the parent of the same sex.

4. Indulgence in alcoholism, with or without sprees, often characterized the rather passive parent of the same sex, usually with acceptance of inferiority rather than rebellious or hateful reaction to criticism by the mate.

The essence of the background seems, in a phrase, to be a prolonged situation in which the significant persons give to the growing child incoherent patterns for adaptation. The love, affection and warmth which are necessary for the development of a stable center of personality are never available. How great a defect one unloving parent may cause is well known, but in most patients one sees that a great deal of damage has been prevented by warm influences coming from the other parent. In the case of patients with amphithymia, however, there has been little or no love, and the patient's grasp on the world of personal realities has been restricted to technics for securing attention, tolerance, none too trustworthy incidents of praise and all too demanding encouragement.

The child comes to be possessed of the most profound insecurity, the absence of love. The need is covered by technics for making successful impressions—impressions which must be disappointing to other persons because there is no ability to respond to warmth and affection. The need is manifested throughout subsequent life in the extreme swings of mood which are called out by thwarting, disappointments, failures and rebuffs. These excursions of mood manifest unrefined destructive impulses directed toward the person concerned, for there is no restricting experience of durable personal intimacy, and only rudimentary sentiments concerning other persons.

The specific deficiency in appraising the reality of other persons must be seen to go with an inadequate formulation of self. The reflected appraisals from the persons significant in the early years have been so unstable and inconsistent that it is simply natural that the patient unhesitatingly adopts any attributes which seem suited to an occasion, with an innocent neglect of any performance to make good the promises. This same factor is responsible for the so-called pathologic lying that frequently characterizes these patients. Its effects on the therapeutic situation will be discussed presently. This factor also is responsible for the luxuriant fantasy which complicates most of these patients' interpersonal relations; in love they have often found perfection, and being incapable of learning from experience each time they are in love they find perfection.

As these persons are without durable reference frames, their attempts at adjustment to others often appear deliberately fraudulent. As a

matter of fact, some of these patients would be perfect "confidence men" if they were able to carry out long term plans. They often intrigue others into contributing in various fashions to their activities, but they generally wear out their welcome soon because of the schematic character of their plans and performances.

PROBLEMS IN TREATMENT

In amphithymic patients one encounters unusual problems in treatment, and they require certain expedients which are not needed in dealing with patients of any other type. This is the direct result in part of the feeble self-organization and formulation of personal reality which have developed in these patients. This factor gives rise to the "fraudulent" personalities which they are at great pains to produce in the more vocal phases of analysis. It underlies the pathologic lying in its more subtle aspects. The defect itself is the result not of repression but of privation. In other words, one cannot expect this aspect to be cured by free association but must focus effort on the actual construction of a correct appraisal of the patient's personal reality. It is often difficult for an analyst in his first contact with a patient of this type to realize how confused the patient is by his incoherent and contradictory reports of his own characteristics.

Of the same genus are the problems resulting from the fluid formulations of other persons by these patients and their protean adapting movements—toward the analyst, for example, in the many rather too sharply focused rôles that are flung over him at the start. By this I mean that the patients, from life-long habit, expand beginning transference phenomena into a wealth of fantasy about the analyst, to such effect that it is almost impossible to sort out the valid transference phenomena from the extravagant imaginings and arabesques. If one considers the identical proceedings in the cases of all the third persons introduced into the analytic material, one realizes how exhausting the early stages of the process must be.

The second fundamental problem in the treatment of these patients is derived from their uncertain contact with reality. I refer to the all-pervading but heavily disguised conviction of inferiority or incompleteness. Here again, the whole experience of the patient's life has been concerned with preventing the appearance of this conviction in awareness. It is not suppressed or repressed; it is, as it were, externally denied in a positive fashion by eternal affirmation of superiority. Unlike the forms of compensation and defensive reactions which one encounters in persons with psychoneurosis, these devices for denial of the patient with amphithymia are fluid in form and are capable of being advanced unhesitatingly in the face of the most effective interpretation.

Being undeterred by the inherent consistency of reality, as other persons know it, these patients progress without embarrassment through the most absurdly contradictory claims to their apparent external complacency. Unlike the patient who feels unworthy of the analyst's attention, these patients always require the analyst to prove worthy of them.

One might think from the statements just made that a therapeutic approach to these patients is, in fact, impossible. Many difficulties in uncovering the dynamics of personality distortions are mitigated by dream analysis. But the analysis of dream processes requires free associations, and it will be observed that the factors thus far discussed are severe prohibitions to free association. There is, however, in the dreams of the person with amphithymia a somewhat fortunate result of the rather schematic contact with personal reality. The feeble self-organization is reflected in the dream life by a paucity of distorting forces. The dreams often approach nearer in manifest content to the conflicts and compromises in the personality than do the dreams of the person with psychoneurosis. I have found that valuable results are often obtained by the interpretation of the manifest content of the dreams which are reported to me, and this has seemed to be the actual entering wedge of the therapeutic process in most cases.

As an indirect result of the feeble self-organization, there is a great variety of incoherent and contradictory statements, even in the course of a single hour. Also, interpretive efforts undergo many vicissitudes, in addition to the almost inevitable denial of their validity with which they are greeted, because of the basic feeling of inferiority and inability of the patient to tolerate the feeling that any one "has anything on" him. The analyst must have always in mind the fact that these patients have an extreme need for demonstrating superior uniqueness, self-sufficiency, priority, etc., and that these needs, in turn, require of the analyst a rigorous holding to the psychoanalytic objective integrity. Of all one's patients these are the persons with whom one must be scrupulously honest and frank in all one's appraisals—needless to say, without any sadistic response to the continued onslaught of the patient. Here also there are subtle demands for conformity with the transference identifications, which, it will be remembered, are essentially incoherent and unreliable personalities, useless to the patient for the elaboration of a stronger organization of the self, the prime achievement of cure.

With the drives for demonstrating superiority, these patients invariably manifest a great repugnance to resuming responsibilities. The resistance to progress in the analysis often expresses itself frankly, if nominally, in a statement of unwillingness to take on any of the responsibilities and conformities of adult social life. Not only is this the case, but the patients often seek actively for types of dependence and irresponsibility they have not previously been able to achieve. This may

take the form of a long-continued campaign to bring about commitment to a sanatorium, placement under supervision or authorization to abandon some responsibilities which they are carrying. Much more dangerous is the insidious pressure to become a responsibility and a dependent of the analyst, to be advised and governed by him.

The last-mentioned characteristic of the patients, when their hostility has become thoroughly focused on the analyst, may take a form which I have called the "albatross motive." The patient seeks to turn the table on the analyst, saying in principle: "I shall ruin your reputation; I shall be a failure to afflict you the rest of your life." In a way, all these movements are to be regarded as steps in the process of breaking up incoherent identifications and establishing durable attitudes in the relation of the self to others. Shot through as they are, however, with the desolation and hopeless rage of the unnumbered disappointments which the child has undergone, dealing with these transference phenomena is exhausting alike to the patience and to the optimism of the analyst. One must take courage from the realization that in a personality which has had little or no experience with durable, powerful integrating motives a consistent and rather well directed hatred is definitely an improvement and a promise of improving integration, if the analyst can survive.

This brings me easily to analogies to the manic-depressive depression which often preoccupy one with prognostic speculations. I refer to the danger of self-destruction of the patient and other implications of threats of suicide. It is no news to physicians well trained in psychiatry that preoccupations with self-destruction and threats of this kind are always complex problems. If my discussion of these patients has been followed, the additional complications which arise from their anomalous contact with reality will be seen. They are, of course, different from those of the patient with schizophrenia, who may kill himself accidentally, as it were, in the pursuit of a psychotic device for salvation. They are also different from those of the truly depressed patient, who is uniformly and consistently preoccupied with the advisability of ceasing to exist. Briefly, these patients are rarely genuinely suicidal, and then only briefly so. The personality organization does not support the profound motives for revenge or the elaborate expiatory fantastic or cosmic processes that appear in the psychoses. The will to live is strong; real suicidal attempts are rare and probably always primarily directed to obtaining more advantages, such as institutional care.

I wish that I could say that one need never be concerned by threats of suicide or other forms of behavior which seem to demand institutional care. There are no limits to the degree to which these patients may deviate from approximate conventional behavior under pressure of the

environment, including the analysis. In general, however, it is unfortunate if recourse to supervision has to be had. Of course, a great deal concerning these patients is unfortunate, and the psychiatrist is confronted in every case with the limits to which his effort can reasonably be expected to extend.

PROGNOSIS

It is obvious that only a measured optimism can be expressed as to the outcome of treatment in any of these patients. Obviously, a history of successful approximation to the conventional moods in business or social life or in the pursuit of an education is more encouraging than its absence. The more frankly and obviously the patient is psychopathic, the more clear it is that a therapeutic effort will take almost as long as the patient has lived if it is to give him really adequate interpersonal relationship. Age and real restrictions imposed by life commitments of the patient are important considerations in predicting results. At best, these patients require a long and singularly difficult analysis, often with considerable attention by the analyst to the adjusting of external situations to support the slowly improving adaptive capacities.

CONCLUSION

It has appeared that swings of mood are not all related to the manic-depressive psychosis. In addition to its homolog the cyclothymic personality, there appears to be another type in which these phenomena are shown but in which the patients differ sufficiently in their dynamics, life pattern and course in psychoanalysis. I have called this type the amphithymic personality and have attempted to delineate its distinguishing characteristics. The condition of the patients falls into the clinical entity of the psychopathic personality, but the amphithymic patients by no means constitute all the patients whose condition belongs in that classification. It seems that the person with amphithymia is the product of a rather definite pattern of family life. He is often amenable to psychoanalytic therapy, but the cure of the deviation is difficult of achievement and peculiarly demanding of the psychoanalyst.

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CONSTITUTIONAL DIFFERENCES BETWEEN DETERIORATED AND NONDETERIORATED PATIENTS WITH EPILEPSY

I. STIGMAS OF DEGENERACY

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For a few years prior to 1927 one of us (H. A. P.) had the opportunity to observe patients with epilepsy in a state hospital. These patients presented characteristic changes in personality: loss of interest, practical inefficiency, excessive religious devotion, virtuous posturing, failure of memory, irritability, egocentricity, disorders of behavior, inactivity, vanity and boastfulness—changes typical of the textbook description of deterioration associated with epilepsy. For a few years after 1927 the same one of us (H. A. P.) was assistant to Dr. Hugh T. Patrick in his private practice and observed under the guidance of this master patients with epilepsy who applied to him for treatment. These presented an entirely different picture, one in which the changes in personality just mentioned were absent. These patients were making their way in their own social, economic, business, artistic and professional worlds with the same ease and grace as their fellowmen; not a few of them came to occupy positions of distinction and responsibility. This experience led to a communication [1] in which it was shown that of 304 patients who had had epilepsy for over six years, and in some instances for decades, only 6.5 per cent showed evidences of deterioration. In this communication it was also pointed out that the patients who did not show deterioration differed in certain particulars from those who did. The disease began later; the patients had fewer seizures and more and longer remissions, and neuropathy was less evident in the stock. In another communication [2] a second difference between the two groups was pointed out, namely, that epilepsy is less prevalent in the children

From the Department of Nervous and Mental Diseases, the Northwestern University Medical School and the Minnie Frances Kleman Memorial Fund.

1. Paskind, H. A.: Extramural Patients with Epilepsy, with Special Reference to the Frequent Absence of Deterioration, Arch. Neurol. & Psychiat. **28**:370 (Aug.) 1932.

2. Paskind, H. A., and Brown, Meyer: Frequency of Epilepsy in Offspring of Persons with Epilepsy, Arch. Neurol. & Psychiat., this issue, p. 1045.

of patients without deterioration than in those of institutionalized patients with deterioration.

Our purpose in the present communication is to report differences in the presence of stigmas of degeneracy [3] in the two groups.

We believe that stigmas of degeneracy may be accepted as constitutional, since they are developmental anomalies which remain unchanged throughout life, except for changes implicit in growth.

We have reviewed the literature to determine what authors have accepted as stigmas of degeneracy and have adopted the criteria of Tredgold,[4] Dana [5] and Church and Peterson [6] as the most modern and comprehensive. These stigmas consist of certain malformations, principally about the head and face, which will be enumerated later in stating the results of our work. From the lists given by these authors we have rejected only the anomalies which we believed might present insuperable subjective difficulties in detection. Regarding the significance of stigmas of degeneracy, most authors are in agreement with Turner,[7] who stated:

> These [stigmas of degeneracy] point to the existence of a latent neuropathic disposition, which may exert a potent influence upon the causation, type of course and treatment of nervous and mental disease. . . . They are of immense value as an index of the intensity, or degree of the hereditary degenerative predisposition. In the most pronounced forms of mental deficiency, such as are seen in imbeciles, idiots and congenital epileptic dements, anatomical variations from the normal are common and often of a pronounced type. In the slighter forms of neuroses, such as neurasthenia, simple melancholia and senile insanity, the stigmata are less frequent and less pronounced than in the more exaggerated conditions above mentioned.

Stigmas of degeneracy have been studied mainly in institutionalized patients. Knecht [8] stated that in 48 persons with epilepsy he found stigmas of degeneracy in 35, or 73 per cent. Eight had lateral deformity

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3. We do not regard the term "stigma of degeneracy" as particularly fortunate; perhaps it should never appear without quotation marks. "Degeneracy" is a word of strong flavor but vague meaning, and stigma carries an unnecessary context of infamy. Perhaps anatomic developmental anomaly would be a more accurate, although less picturesque, term, for that is what stigma of degeneracy implies. However, since the expression is so firmly entrenched in the literature, we have thought best to employ it for the purposes of this communication.

4. Tredgold, A. F.: Mental Deficiency, New York, William Wood & Company, 1920.

5. Dana, C. L.: Textbook of Nervous Diseases, New York, William Wood & Company, 1925.

6. Church, A., and Peterson, F.: Nervous and Mental Diseases, ed. 10, Philadelphia, W. B. Saunders Company, 1923.

7. Turner, W. A.: Epilepsy: A Study of the Idiopathic Disease, London, Macmillan and Company, 1907.

8. Knecht: Ueber die Verbreitung physischer Degeneration bei Verbrechern und die Beziehungen zwischen Degenerationszeichen und Neuropathien, Allg. Ztschr. f. Psychiat. **40**:584, 1883.

of the skull (3 of whom had facial paresis); 2, asymmetry of the head and face; 5, deviation of the axis of the face from the perpendicular; 5, a skull characteristic of hydrocephalus; 12, a "birdlike" facial profile; 2, a small, infantile face; 10, unequal innervation of the sides of the face; 13, anomalies of the ear; 3, abnormal teeth; 2, phimosis; 1, cryptorchidism, and 1, multiple nevi. Sollier [9] reported retardation of the second dentition in 25 per cent of persons with epilepsy. Of 128 patients Féré [10] found asymmetry of the skull in 71 per cent; this condition was found in 55 per cent of subjects used as controls, some of whom had neuropathy. He found difference in the color of the irides in 4 per cent of persons with epilepsy; this he never observed in the normal persons studied as controls. Of 116 patients he found differences in the shade of the irides in 26.7 per cent, inequality of the pupils in 9.48 per cent and eccentricity of the pupils in 4.3 per cent. Of 130 patients he found that 60 per cent had asymmetry of the hands and 64.6 per cent asymmetry of the feet. In 51.5 per cent of 134 persons with epilepsy he found anomalies in the length of the fingers; in 54.3 per cent of 140 patients he observed a tubercle at the angle of the jaw. Clouston [11] stated that he found abnormality of the palate in 80 per cent of persons with epilepsy. Ganter [12] reported that such persons have more anomalies of the iris, ear, teeth, jaws, face, skull and extremities than normal persons. Talbot [13] stated that abnormalities of the palate were found in 76.3 per cent of 55 patients with epilepsy and in 25.3 per cent of 1,000 normal persons. Turner [7] found that of 200 persons with epilepsy 42 per cent had facial asymmetry, 41.5 per cent abnormality of the palate and 23 per cent abnormality of the ears. Charon [14] found abnormality of the palate in 76 per cent in his series. Nöcke [15] reported the presence of torus palatinus in 32.9 per cent of women with epilepsy. Dana [5] stated that stigmas of degeneracy are ten times more frequent in patients with epilepsy than in normal persons; in the former he described the frequent presence of prominence of the occiput, lemurian hypophysis, asymmetry of the pupils and iris, misplaced crown of the scalp, long fingers and greater development of the left side.

9. Sollier, A.: De l'état de la dentition chez les enfants idiots ou arrières, Thèse de Paris, no. 4, 1887.

10. Féré, C.: Les épilepsies et les épileptiques, Paris, Félix Alcan, 1890.

11. Clouston, T. S.: The Neuroses of Development, Edinburgh, Oliver & Boyd, 1891.

12. Ganter, R.: Ueber Degenerationszeichen (von Iris, Ohr, Zähnen, usw.) bei Gesunden, Geisteskranken, Epileptikern und Idioten, Allg. Ztschr. f. Psychiat. **70**:205, 1913.

13. Talbot, E. S.: Irregularities of the Teeth, Philadelphia, S. S. White, 1903.

14. Charon, cited by Church and Peterson.[6]

15. Nöcke, cited by Church and Peterson.[6]

MATERIAL

The subjects utilized for the present study consisted of 79 epileptic patients with deterioration in the Chicago, Elgin and Dixon State Hospitals and 39 patients with no deterioration from the outpatient clinics of the Northwestern University Medical School, the Evanston Hospital and the Michael Reese Hospital. Of the deteriorated patients, 64 were men and 15 women; of the nondeteriorated patients, 28 were males and 11 females. The ages of the deteriorated patients varied from 20 to 76 years, and those of the other group of patients, from 14 to 63 years; in both groups most of the patients were between 20 and 40 years of age. In order to allow time for deterioration to occur, no patient was accepted for the group of those not showing deterioration unless he had had seizures for at least four years; many of these patients had had seizures for decades. In both groups we were careful to exclude persons with defective mental development; in some instances this was done with the aid of psychometric tests; in others, by a study of the educational and vocational histories of the patient. No patient was accepted who had signs of focal neurologic disorder. In order to avoid errors of subjective appraisal, we recorded only the stigmas which were obvious and marked and those that we could easily compare with the descriptions and illustrations of other authors.

RESULTS OF INVESTIGATION

The number of times each stigma occurred in the deteriorated and nondeteriorated patients, together with the rate of occurrence per hundred patients, is shown in table 1. Study of this table shows that all stigmas, with the exception of attachment of the ear lobe, accessory anthelix, partially fused helix and anthelix, unusually prominent upper lip and abnormally high palate, were more common in the deteriorated patients. In most instances these differences were marked, certain stigmas occurring several times as frequently in the deteriorated as in the nondeteriorated patients. In other instances certain anomalies were found in the deteriorated patients that were absent in the nondeteriorated patients.

Table 2 shows the percentage of deteriorated and nondeteriorated patients with stigmas in various parts. Study of this table shows that in all the parts included in the study malformations were more frequent in deteriorated than in nondeteriorated patients. Anomalies of the eye were over twice as common in deteriorated as in nondeteriorated patients, and those of the teeth were over three times as common. Anomalies of the skull were less than one-third as frequent in nondeteriorated patients; those of the nose were one-half as frequent. Defects in palatal structure were almost twice as common in deteriorated as in nondeteriorated subjects.

Table 3 indicates the number and percentage of deteriorated and nondeteriorated patients, respectively, having a given number of stigmas. Study of this table shows that the deteriorated patients were more heavily stigmatized, patients of this group having more defects per person than did the group of nondeteriorated patients. Of the deterio-

TABLE 1.—*Number of Observations of Each Stigma in Deteriorated and Nondeteriorated Patients with Epilepsy, with Rate of Occurrence per Hundred Patients*

Stigma	Deteriorated Patients (79)		Nondeteriorated Patients (39)	
	No. of Observations of Each Stigma	Frequency of Occurrence per Hundred Patients	No. of Observations of Each Stigma	Frequency of Occurrence per Hundred Patients
Asymmetry of eyebrows	7	8.8	1	2.6
Speckled irides	84	106.0	14	36.0
Obliquity (downward and outward) of one palpebral fissure	7	8.8	0	0.0
Obliquity (upward and outward) of one palpebral fissure	8	10.1	0	0.0
Oliquity (downward and outward) of both palpebral fissures	7	8.8	3	7.7
Obliquity (upward and outward) of both palpebral fissures	9	11.4	1	2.6
Asymmetrical palpebral fissures (size, position and direction)	26	33.0	5	13.0
Epicanthus	40	51.0	10	26.0
Axis of ear directed too far backward	13	14.0	0	0.0
Unusually prominent ear	66	83.0	12	31.0
Abnormally large ear lobe	45	57.0	16	41.0
Abnormally small ear lobe	48	61.0	10	26.0
Attached ear lobe	63	80.0	40	103.0
Triangular ear lobe	80	101.0	0	0.0
Triangular ear	2	2.5	0	0.0
Satanic ear	8	10.1	0	0.0
Asymmetry (size, prominence, position, etc.) of ear	42	53.0	4	10.3
Poorly developed helix	47	59.0	4	10.3
Poorly developed anthelix	10	13.0	0	0.0
Poorly developed tragus	1	1.3	0	0.0
Overdeveloped anthelix	26	33.0	0	0.0
Overdeveloped tragus	2	2.5	0	0.0
Overdeveloped antitragus	3	3.8	0	0.0
Accessory anthelix	0	0.0	1	2.6
Tricornuate anthelix	1	1.3	0	0.0
Unicornuate anthelix	1	1.3	0	0.0
Partially fused helix and anthelix	0	0.0	1	2.6
Darwinian tubercle	23	29.0	4	10.3
Abnormally large palate	17	22.0	8	21.0
Abnormally small palate	16	20.0	3	7.7
Abnormally high palate	24	30.0	13	33.0
Abnormally shallow palate	20	25.0	0	0.0
Abnormally wide palate	21	27.0	3	7.7
Abnormally narrow palate	33	42.0	8	21.0
Shelf in palate	5	6.4	0	0.0
Asymmetrical palate	12	15.0	0	0.0
Torus palatinus	10	13.0	0	0.0
Saddle-shaped palate	11	14.0	0	0.0
V-shaped palate	9	11.0	2	5.1
Irregular teeth	26*	41.0*	4†	12.0†
Poorly formed teeth	9*	14.0*	1†	2.9†
Abnormally large teeth	1*	1.6*	0†	0.0†
Abnormally small teeth	7*	11.0*	1†	2.9†
Abnormally large tongue	26	33.0	10	26.0
Abnormally small tongue	4	5.1	1	2.6
Unusually prominent median fissure of tongue	5	6.4	1	2.6
Lobulation of tongue	2	2.5	0	0.0
Abnormally increased markings of tongue	12	15.0	3	7.7
Unusually prominent upper lip	9	11.0	5	13.0
Unusually prominent lower lip	12	15.0	4	10.3
Open mouth	18	23.0	1	2.6
Eversion of lower lip (membranous portion)	20	25.0	2	5.1
Lateral deviation of nose	6	7.5	0	0.0
Cleft above nose	1	1.3	0	0.0
Nostrils directed forward	18	23.0	6	15.0

TABLE 1.—*Number of Observations of Each Stigma in Deteriorated and Nondeteriorated Patients with Epilepsy, with Rate of Occurrence per Hundred Patients—Continued*

Stigma	Deteriorated Patients (79)		Nondeteriorated Patients (39)	
	No. of Observations of Each Stigma	Frequency of Occurrence per Hundred Patients	No. of Observations of Each Stigma	Frequency of Occurrence per Hundred Patients
Facial asymmetry	46	58.0	7	18.0
Prognathism	14	17.0	4	10.3
Unusually prominent cranial bosses (temporoparietal, mastoid and frontal)	3	3.8	0	0.0
Abnormally large supra-orbital ridges	8	10.1	2	5.1
Polydactylism	0	0.0	0	0.0
Syndactylism	5	6.4	1	2.6
Abnormal separation of first and second toes	6	7.5	0	0.0
Digital deformity	9	11.0	1	2.6
Tumefactions of skin‡	2	2.5	0	0.0

* Based on a total of 64 patients.
† Based on a total of 34 patients.
‡ No histologic studies of these cutaneous tumefactions were made. In both patients the lesions were similar to those seen in Recklinghausen's disease. They consisted of myriad nodules, ranging in size from that of a pinhead to that of an olive and were of purplish color and freely movable. They were not tender and were most common on the trunk and less so on the face and extremities.

TABLE 2.—*Number of Deteriorated and Nondeteriorated Patients Showing Stigmas of Various Parts of the Body*

Stigmatized Part	Deteriorated Patients (79), Percentage	Nondeteriorated Patients (39), Percentage
Appendages of eye*	83.0	41.0
Ear	95.0	79.0
Palate	77.0	46.0
Teeth	52.0†	15.0‡
Tongue	47.0	33.0
Lips	42.0	23.0
Nose	32.0	15.0
Facial asymmetry	58.0	18.0
Prognathism	17.0	10.0
Skull	8.8	2.6
Fingers and toes	13.0	2.6
Skin	2.5	0.0

* These included the iris.
† Based on a total of 64 patients.
‡ Based on a total of 34 patients.

TABLE 3.—*Number and Percentage of Deteriorated and Nondeteriorated Patients Having a Given Number of Stigmas*

Number of Stigmas	Deteriorated Patients (79)		Nondeteriorated Patients (39)	
	No. of Patients	Percentage	No. of Patients	Percentage
0-2	1	1.3	8	21.0
3-5	4	5.1	11	28.0
6-8	11	14.0	13	33.0
9-11	18	23.0	7	18.0
12-14	20	25.0	0	0.0
15-17	13	16.5	0	0.0
18-20	8	10.0	0	0.0
Over 20	4	5.1	0	0.0

rated patients 56.6 per cent had twelve or more stigmas; in the group of nondeteriorated patients such profuse stigmatization did not occur in a single instance.

COMMENT

From the preceding studies we are convinced that stigmas of degeneracy occur much more frequently in deteriorated than in nondeteriorated patients with epilepsy.

The significance of these differences remains to be discussed. Stigmas of degeneracy represent essentially imperfect structural development. It seems reasonable to us that the brain may not be exempt from such imperfections and that there may be a correlation between visible stigmas of degeneration and the presence of developmental anomalies of the brain. Higier [16] expressed the belief that there is a correlation between stigmas of degeneracy and defects in cerebral development. Bittorf [17] reasoned that this is to be expected, for most stigmas of degeneration are found in ectodermal structures and the brain is also an ectodermal organ.

Further, several authors have described developmental defects in the brains of persons with epilepsy. Thus, J. Turner [18] stated that in 27 of 35 cases he observed embryonic forms of Betz cells and in 7 of 13 cases defective development of the posterior columns and that all the brains showed subcortical ganglion cells. In a subsequent paper he [19] reported that in 27 of 36 brains of persons with epilepsy he observed subcortical ganglion cells. W. A. Turner [7] reported a reduction in the number of cells in the outer layers of the cortex, especially in the second and third layers, embryonic types of Betz cells, persistence of nerve cells in the white matter and abnormal arrangement of tangential fibers. Kraepelin [20] described fetal types of cells in the molecular layer of the cortex in association with epilepsy and pointed out that this is a developmental anomaly in which these cells fail to enter the lower layers as they do in normal adults. Wohlwill [21] stated that developmental anomalies are common in the brains of persons with epilepsy. He described forms of heterotopia, Cajal fetal cells and poor differentiation of cortical cell layers and of the cortex from the underlying white matter. Bauer [22]

16. Higier, H.: Pathologie der angeborenen familiären und hereditären Krankheiten, speziell der Nerven- und Geisteskrankheiten, Arch. f. Psychiat. **48**:41, 1911.

17. Bittorf, quoted by Higier.[16]

18. Turner, J.: The Pathology of Epilepsy, Brit. M. J. **1**:496, 1906.

19. Turner, J.: The Pathological Anatomy and Pathology of Epilepsy, J. Ment. Sc. **53**:1, 1907.

20. Kraepelin, E.: Psychiatrie, Leipzig, J. A. Barth, 1913, vol. 3.

21. Wohlwill, F.: Entwicklngsstörungen des Gehirns und Epilepsie, Ztschr. f. d. ges. Neurol. u. Psychiat. **33**:261, 1916.

22. Bauer, J.: Konstitution und Nervensystem, Ztschr. f. d. ges. Neurol. u. Psychiat. **15**:161 and 337, 1918.

reported the presence of Cajal fetal cells, misplaced ganglion cells, indistinct separation of cortical layers, abnormal forms of nerve cells, heterotopia and microgyria. Spielmeyer [23] stated that in cases of so-called genuine epilepsy developmental anomalies of the brain are frequent and that they play an essential part in the pathologic picture in the majority of cases. He stated that Wohlwill pointed out correctly that these developmental anomalies are not mere coincidence.

All the observations described, however, were reported in cases of institutionalized patients with deterioration. After a thorough review of the literature we have not found a single report of necropsy in the case of an extramural nondeteriorated patient with epilepsy. It seems reasonable to believe that if there is a correlation between visible stigmas of degeneracy (somatic developmental anomalies) and cerebral developmental anomalies, one would expect to find fewer developmental defects in the brains of nondeteriorated patients, since they have fewer somatic stigmas of degeneracy. This even suggests that an essential difference between institutionalized deteriorated and extramural nondeteriorated patients with epilepsy may be the more frequent presence of cerebral developmental anomalies in the patients with deterioration.

SUMMARY AND CONCLUSIONS

Seventy-nine deteriorated and 39 nondeteriorated patients with epilepsy were studied for the presence of stigmas of degeneracy. These were much more common in the deteriorated patients. It seems reasonable to believe that there is a correlation between stigmas of degeneracy (somatic developmental anomalies) and cerebral developmental anomalies. This suggests that an essential difference between institutionalized deteriorated and extramural nondeteriorated patients with epilepsy may be the more frequent presence of cerebral developmental anomalies in the patients with deterioration.

Dr. Charles F. Read, Dr. Warren G. Murray and Dr. Edward Dombrowski, the managing officers of the Elgin State Hospital, the Dixon State Hospital and the Chicago State Hospital, respectively, gave permission to study these patients and cooperated in the furtherance of this study.

23. Spielmeyer, W.: Der gegenwärtige Stand der Epilepsieforschung, Ztschr. f. d. ges. Neurol. u. Psychiat. **89**:360, 1924.

FREQUENCY OF EPILEPSY IN OFFSPRING OF PERSONS WITH EPILEPSY

WITH SPECIAL REFERENCE TO DIFFERENCES BETWEEN INSTITUTIONAL AND EXTRAMURAL PATIENTS

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Studies of the incidence of epilepsy in the offspring of persons with epilepsy have hitherto been made almost exclusively with institutionalized patients. Such patients, however, are a highly selected group, for in addition to the presence of seizures there have occurred mental changes, principally deterioration and clouded states, which necessitated hospitalization. The great mass of persons with epilepsy who are not institutionalized have not been included in such studies because writers in institutions have had no contact with them. Some characteristics of extramural patients have already been indicated by one of us (H. A. P.);[1] namely, only 6.5 per cent show deterioration, the remainder adjusting themselves to life as well as their fellowmen; they come of a stock with a lighter burden of neuropathy; they have fewer seizures; the onset of the disease is later, and the disease has more and longer remissions.

Although no exact statistics regarding the proportion of institutionalized to extramural patients with epilepsy can be cited, we are convinced that the latter greatly outnumber the former and that their exclusion from studies of epilepsy has given rise to reports which deal not with the broader aspects of the disease but only with the phases encountered in epileptic patients with psychosis.

The purpose of this communication is to report the incidence of epilepsy in the children of a group of extramural patients who did not show deterioration and to compare these data with those for the classic patients with deterioration given in the literature.

In 1826 Bouchet and Cazauvieilh[2] reported that of 58 children born of 14 mothers with epilepsy, 3.8 per cent had epilepsy. Dusart[3]

From the Department of Nervous and Mental Diseases, the Northwestern University Medical School and the Minnie Frances Kleman Memorial Fund.

1. Paskind, H. A.: Extramural Patients with Epilepsy, with Special Reference to the Frequent Absence of Deterioration, Arch. Neurol. & Psychiat. **28**:370 (Aug.) 1932.
2. Bouchet, C., and Cazauvieilh, J., quoted by Féré.[8]
3. Dusart, L. O., quoted by Féré.[8]

observed that 36 of 70 children of parents with epilepsy died in convulsions. Tardieu [4] stated that of 72 such children 33 had convulsions and 21 died during seizures. Voisin [5] studied the incidence of epilepsy in 35 children of families in which the disease had occurred; he found that 16, or 45.7 per cent, had epilepsy or died of convulsions. Echeverría [6] studied 553 children of 136 patients with epilepsy; 195 of these died of infantile convulsions, and 78, or 14.1 per cent, had epilepsy. Foville [7] stated that 25 per cent of the issue of patients with epilepsy have the disease. Féré [8] reported that 30, or 8.5 per cent, of 352 descendants of 120 such patients had epilepsy and 87, or 24.7 per cent, infantile convulsions. Starr [9] found that 17, or 0.8 per cent, of 2,000 children with one parent suffering from epilepsy had the disease. Collins [10] reported that of 197 such children 10, or 5 per cent, died of convulsions and 5, or 2.5 per cent, had epilepsy. Thom [11] concluded that the incidence of epilepsy in the issue of persons with the disease is a little less than 2 per cent. Van Londen [12] found that of 316 children born of 119 affected parents, 15, or 4.7 per cent, had convulsions. Hoffmann [13] found the disease in 3, or 11.11 per cent, of 27 adult offspring of 8 parents with epilepsy. Krisch [14] stated that 10.34 per cent of children of affected parents have epilepsy. Of 443 such children, Burr [15] found epilepsy in 6, or 1.3 per cent, and infantile convulsions in 9, or 2.03 per cent. Coberis [16] stated that 3 of 72 children of parents with epilepsy died in convulsions. Brain [17] reported that of 56 children

4. Tardieu, J., quoted by Echeverría.[6]

5. Voisin, A.: Ann. méd. psychol. **12**:114, 1868.

6. Echeverría, M. G.: Marriage and the Hereditariness of Epilepsy, J. Ment. Sc. **26**:346, 1881.

7. Foville, A., quoted by Déjerine, J.: L'hérédité dans les maladies du système nerveux, Paris, Asselin & Houzeau, 1886.

8. Féré, C.: Les épilepsies et les épileptiques, Paris, Félix Alcan, 1890.

9. Starr, M. A.: Organic and Functional Nervous Diseases, Philadelphia, Lea & Febiger, 1913.

10. Collins, M. A.: The Hereditary Transmission of Epilepsy, Epilepsia **4**:365, 1914.

11. Thom, D. A.: A Second Note on the Frequency of Epilepsy in the Offspring of Epileptics, Boston M. & S. J. **175**:599, 1916.

12. van Londen, D. M.: Should an Epileptic Marry? Nederl. tijdschr. v. geneesk. **65**:2048, 1921.

13. Hoffmann, H.: Die Nachkommenschaft bei endogenen Psychosen, Berlin, Julius Springer, 1921.

14. Krisch, H.: Die biologische Einteilung der Epilepsien, Monatschr. f. Psychiat. u. Neurol. **52**:312, 1922.

15. Burr, C. W.: Heredity in Epilepsy: A Study of One Thousand Four Hundred and Forty-Four Cases, Arch. Neurol. & Psychiat. **7**:721 (June) 1922.

16. Coberis, G.: L'ereditaria nell'epilepsia, Quaderni di psichiat. **12**:4, 1925; abstr., Zentralbl. f. d. ges. Neurol. u. Psychiat. **41**:242, 1925.

17. Brain, W. R.: Inheritance of Epilepsy, Quart. J. Med. **19**:299, 1926.

born of 24 persons with epilepsy, 3, or 5.3 per cent, were similarly afflicted. Pilcz [18] found 3 patients (1.9 per cent) with epilepsy in a group of 153 children born of 144 mothers with the disease. Küenzi [19] found that 4, or 3 per cent, of 132 persons over 20 years of age with an affected parent had epilepsy. Stein [20] reported that of 267 children of 87 parents with epilepsy, 2.2 per cent had the same disorder. He cited Lennox and Cobb as having found that 1.1 per cent of children of extramural patients with epilepsy had this disorder. Boening and Konstantinu [21] reported that they observed convulsions in 8 of 94 children of parents with epilepsy, or in 8.5 per cent.

These ratios, it must be remembered, were derived in almost all instances from studies of institutionalized deteriorated patients. Further, the data are somewhat obscured by the fact that some authors did not exclude children of parents who were fundamentally feebleminded and in whom the seizures were a manifestation of a gross developmental defect of the brain. This may account partially for the wide variations in the figures of the authors cited.

MATERIAL

The material used for the present study consisted of the records of 162 parents with epilepsy from the private practice of Dr. Hugh T. Patrick. The differences which these patients present from the institutionalized deteriorated patients have already been pointed out. The records contained data on 342 living children and on 28 children who had died.

RESULTS OF INVESTIGATION

Of the 342 living children, the age was recorded for 147: Forty-two were under 5 years of age; 36, from 6 to 10 years; 31, from 11 to 15 years; 14, from 16 to 20 years; 15, from 21 to 25 years, and 9, over 25 years of age. For 195 of the offspring there was no record of age, but it was thought that the length of time that the affected parent had been married might afford a clue as to the age of the issue. The parents had been married less than five years in 10 instances, from six to ten

18. Pilcz, A.: Die weiteren Lebensschicksall von Kindern-welche während des Bestehens einer Mütterlichen Geist-oder Nerven-krankheit geboren worden sein, Jahrb. f. Psychiat. u. Neurol. **46**:153, 1929.

19. Küenzi, F.: Ueber das Wiederauftreten von Epilepsie unter den Nachkommen von Epileptikern, Monatschr. f. Psychiat. u. Neurol. **72**:245, 1929.

20. Stein, C.: Hereditary Factors in Epilepsy: Comparative Study of 1,000 Institutional Epileptics and 1,115 Non-Epileptic Controls, Am. J. Psychiat. **12**: 989 (March) 1933.

21. Boening, H., and Konstantinu, T.: Encephalographische und erbbiologische Untersuchungen an genuinen Epileptikern, Arch. f. Psychiat. **100**:171, 1933.

years in 17 instances and from eleven to fifteen years in 45 instances. Forty of the subjects had parents who had been married from sixteen to twenty years; 14 from twenty-one to twenty-five years, and 65, over twenty-five years. In 4 records the duration of marriage was not recorded. It appears from these data that many of the children whose ages were not recorded must have been at least in late youth or early middle life.

Of the present series of 342 children born of parents with epilepsy, the disease was found in only 1, or 0.29 per cent. Infantile convulsions occurred in 6, or 1.7 per cent.

This ratio, obtained from a study of the offspring of nondeteriorated, extramural patients, is markedly lower than the similar ratio for the offspring of institutionalized deteriorated patients as given in the literature.

Our statistics are not altered by the possibility of epilepsy in the children who died. Neither epilepsy nor infantile convulsions occurred in any of these children. The causes of death in this group were: meningitis, 3; tuberculosis, diabetes, influenza, rheumatism, smallpox and pneumonia, 1 each; cholera infantum, 3; diphtheria and accident, 2 each, and an unknown agent, 12.

CONCLUSIONS

Of 342 children born of 163 extramural patients with epilepsy, the disease was found in 0.29 per cent and infantile convulsions in 1.7 per cent. These figures are markedly lower than those given in the literature for deteriorated institutionalized patients.

EMOTIONAL FACTORS IN MENTAL RETARDATION

A READING PROBLEM

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The psychiatry of childhood differs from the psychiatry of adulthood in several important particulars. Although one cannot safely assume that the mind of the new-born infant is entirely a blank, from the conventional point of view it is nearly that, whereas the mind of the adult has been exposed to the influence of convention throughout childhood and hence is bound to be biased. Perhaps the other reasons for the differences between these two aspects of psychiatry depend on this fundamental fact. However, there are mechanisms whereby these differences express themselves. One important point of view is associated with and dependent on the rate at which a child constantly learns. Such a process, of course, means constant change. This is particularly important when one realizes how severe man is toward his mistakes. A person can be right many times and have no particular emotional reaction therefrom, but if he is wrong once he may be caught, and this particular event may be the center of much of his subsequent living. The struggle is constantly toward betterment. Humility is praiseworthy; boasting is objectionable. "We are all miserable sinners." Such phrases are expressions of the socially acceptable or desirable attitude. To express this matter in another way, it is said that a chimpanzee with the frontal portion of the brain intact tears his hair, screams his curses and falls in a rage when he makes a mistake in the course of training. However, a chimpanzee in which the frontal part of the brain has been amputated takes his mistakes with equanimity. Man has the frontal lobe of the brain not only intact but much more developed than that in the monkey, and when he makes mistakes he can turn not from one twentieth to one fortieth of his central nervous system against himself but almost a fifth or a tenth.

It is in the process of learning that one comes to know one's mistakes. In the first fifteen years of life one learns so rapidly that a sense of these mistakes is present, if not much of the time, at least at frequent intervals. Also, the things one learns in the earlier years are of much greater significance than most that is learned in later life. These significant things can be roughly divided into two general categories: knowledge of one's body and a feeling of right and wrong.

The former class may be thought of as tinged with instinct—perhaps arising from instinct. At least, it is based on the possession of a body and a need to know how to use it. This need is a part of instinct in the sense that the various organs demand function and that, in the case of organs in contact with the outside world, they have to learn to function in relationship with that world. The eyes must see, the ears hear, the mouth taste and absorb, the throat swallow, the sphincters allow expulsion, the hands handle, the legs locomote and, after a period of from twelve to fourteen years, the genitals generate.

This list does not include the function of speech. In a sense, it is the most important of all the functions that enter into relation with the outside world. At least, from the point of view of learning its importance is supreme. First, the mechanics of speech must be learned. The number of stammerers and stutterers, lispers and children with other failures or inadequacies in the efficient production of sounds evidence the difficulties of this fundamental type of learning. Second, words must be learned. One might well say that learning in the academic sense is based on words. A few essentials must be acquired in relation to words. The sound of the word must be learned, and how it looks: Ears and eyes must function with it. It must be pronounced: The speech mechanism must function with it. Its meaning must be understood: The mind must function with it. Last, but no less important, its emotional significance must be learned: Is it right or is it wrong?

In the foregoing sense, words and their use partake of or enter into both the general classes into which the significant learning of childhood is divided. They bring into activity the eyes, ears, brain and speech mechanisms—also eventually, of course, the writing mechanism. Their use demands a learning of definition, and about their use are erected the mazes of right and wrong, good and bad, desirable and dangerous.

Definition is learned by word of mouth, by being told and by reading. In being told, definition is acquired through some one else; in reading, definition or meaning is acquired through one's own efforts. The process is something like this: A general sense of the meaning of the word is conveyed by its appearance; this is added to by its context, and, eventually, when it is met in different situations the meaning gradually becomes increasingly defined. The sense of definition is clinched by correct pronunciation and exact spelling.

For several years cases of difficulty with reading have been reported. Children have been studied who seemed to have a specific difficulty with the recognition of printed or written words.

Twenty-five years ago I saw a man, aged 35, who was offered a good executive position in the leading industrial institution in his part

of the country. He was unable to make up his mind to accept it because the duties demanded his reading a report to the directors once a month. This prospect filled him with dread. In three or four visits what seemed to be the origin of his difficulty was discovered. While reading aloud in the third reader at school, at the age of 10 or 11, he had slightly mispronounced the word "shooting." As he did so, a boy in the seat beside which he was standing looked up at him and said "shiting," the first "i" being sounded as in "side." He was unable to go on and sat down in confusion. Through some hook or crook, he managed to be excused from further reading aloud in school and had never read aloud in public since.

This case suggested the element of danger that exists in words. Other experiences have strengthened the impression that difficulties with words might have an emotional element of the same general nature as was at work in the case cited. If the 10 year old schoolboy had not known the word that was the next logical sound after "shooting" and "shiting," he would have had no trouble. Children delight in the clang of resemblances of words (the origin of rhymed verse). They make sounds that suggest each other. The sounds may be words. There seems to be particular enjoyment when in chanting such sounds a word is recognizable. If the word is near one of the taboo words the enjoyment is intensified. Because the boy just mentioned did know the word, he had felt dread and chagrin many times through twenty-five years of his life and at 35 was seriously considering refusal of advancement because he could not stand on his feet and read aloud.

In recent years the work of Orton has directed attention to the element of handedness and dominance of a hemisphere.

The boy whose story forms the basis of this report was left handed, and some efforts had been made to induce him to use his right hand, first in eating and then in writing. These efforts had been recognized as unfortunate by the mother and were not persisted in. I could not be sure from what she told me how strenuous the effort had been. The fact that she wanted me to know that she was right in her attitude toward this problem fitted so nicely into my theory of an important element of the difficulty that it seemed to strengthen the basis of my theory. She said that the boy had been "forced against her wishes to write with his right hand." It seemed to me that she stressed the pressure placed on the boy by the teacher so as to lessen her own sense of responsibility in the cause of the difficulty. She had been a school teacher and had married a man of superior education, who had died when the patient was 5 years old. She was left in difficulties, and shortly before I first saw the boy, she had married a man with only a minimum of schooling. She left him after two years because he was so severe with the boy.

During the four years in which I studied this boy the mother had a great deal of trouble. One result of her difficulties was a determination, and she was a very determined woman, that the boy should be right, that he should know what he ought to know and not what he ought not. In the pages that follow, an attempt is made to show how this pressure interfered with his efforts at learning.

The boy, aged 9 years, was brought to the clinic because of difficulty with reading. In the first visit he told of being scared by the reading teacher "when she gets mad." He had been sent home from school and "out in the hall," and he was afraid that the janitor would take him "and do something" to him. He told of seeing tigers in his dreams.

When first seen the boy was in the third grade, but because of difficulties in reading and general lack of cooperation, he was put back into the room for subnormal children. The mother expressed the belief that at 10 years of age he did not "read as well as he did at 5." "He doesn't know the word 'was.'"

At this time the boy said of his stepfather: "If he hits me I won't do anything. If he doesn't hit me I'll try to mind. He hits me when I do a little thing. I don't care if he hits me when I do a bad thing. If he hits me I'm going to run away." Then, in speaking of the teacher, he said in the same highly resentful tone of voice: "She goes out almost every day; another teacher comes in and tells us things, and she won't even write them out." When I said: "Well, you couldn't read it, anyway," he answered: "Sure, I could." When I asked him to write his name and he wrote it with his left hand, I said: "I suppose it scared you when the teacher made you write with your right hand." "Yes," he said, "and I won't do it, either."

He was not seen for almost a year. As soon as he came into my room at the clinic, he wrote his name on the blackboard. He wrote so poorly that I said: "Let's see you write your best." He then wrote much better, with such ease that I had the distinct impression that the first writing was done badly on purpose; so I said: "You can write fairly well when you want to, but usually you don't want to. I guess you don't care to learn." To this his reply was: "I get mad and lose my temper." Then this was followed by talk of how "mad" he got at his teacher and at his mother and, finally, of how his stepfather whipped him. Then he told of a dream of seeing a woman have her back broken and of how robbers "didn't believe a woman who said she didn't know the combination of the safe; so they killed her." In the conversation that followed he told me of reading in the papers of robbers breaking a woman's back because she would not tell them the combination of the safe. I expressed doubt of his story. He became angry, and when the mother was called in he greeted her with: "He tried to tell me there was no robbery at the Rainbow Gardens," in a most accusatory, truculent tone. The mother then said that the patient really saw a girl tied up, who asked him to call the police, but that she was released before the police came. Then she told of the patient taking 15 cents from her purse and of his denying it but finally admitting the act when she found the money in his pocket.

One, therefore, can understand the boy's anger. He pieced together what was read, what he had seen in a show and what really had happened and made a terrible story of the torture of a woman. He accused me of denying what he said had happened and insisted in a loud voice that I said there was no robbery; then, finally, the mother told of an immediate source of guilt, the stealing from her purse. Obviously, to become angry with some one else and to divert attention from oneself are to attempt to divert criticism.

At the next visit he told of becoming angry with his teacher.

Doctor: "Why?"

Patient: "I thought she was wrong, but I was wrong."

Doctor: "Why?"

Patient: "She said I didn't learn my lesson, and I didn't."

At this point he went to the blackboard on which I had written the name "Martin" and under it

$$\begin{array}{r} 17 \\ 12 \\ \hline 32 \end{array}$$

This had been written to test the reactions of a preceding patient. While talking to me, he leaned against the wall, put a small 9 just above the 2 of the 32 and a 2 above the 3, thus correcting me and showing that he could do such addition if he wanted to and nobody told him to. This was done in a thoroughly casual manner, without comment. I then asked him to rub out the whole thing which he did. Then, as we played "catch," I asked him to spell "Martin."

Patient: "M-a-r-t-a-n" (with a slight but definite accentuation of the second "a").

Doctor: "No."

Patient: "M-a-r-t- (slight pause) o-n."

Doctor: "No."

Patient: "M-a-r-t-e-n" (with almost a complete stop after the "t" and a grin as he said "e." I joined in the laugh, and then he spelled "M-a-r-t-i-" quickly, looked at me archly and then exploded with an "e."

Doctor: "So that's the way you try to fool the teacher?"

Patient: (Accepts with a giggle.) "I get mixed up."

The doctor asked the patient if he would return, and he responded: "Maybe tomorrow."

The impression this visit made on me was that this boy could be smart if he wanted to be. One of the times at which he might be expected to want to be smart was when he could correct an adult. On my blackboard, in figures and adult writing, were a word and a problem in simple addition, the latter done incorrectly. Without any hint from me but after glancing at it two or three times, he corrected the mistake in addition. It required no painsaking attention. It was done as we were playing "catch" and talking. Then, the way that he misspelled "Martin" seemed purposeful. He definitely accentuated the mistake in the first attempt. This emphasis was distinctly greater in the second trial, and in the third it was so obvious that I had to acknowledge it with a laugh, in which he joined. In the fourth and final attempt the rapid, correct spelling up to the last letter, the provocative, expectant pause and the explosive "e" seemed to show that he could spell correctly if he wanted to but that it was more satisfactory to fool or thwart the expectant adult. That he ended with an "e" might be thought to show that he knew that the word could be spelled either with an "i" or an "e," though this is farther than I care to go.

Five weeks later he returned. After he had told me he thought of "scary things that would kill" him after he went to bed, I returned to the spelling of "Martin."

Doctor: "We played that game before with 'Martin.' You spelled 'M-a-r-t'."

Patient: "a-n."

Doctor: "Then what?"

Patient: "I don't see how you can remember that. I can't."

Doctor: "Don't remember! There was something about addition on the board."

Patient: "Yes, and I erased it. Here, I'll put it on the board:

$$\begin{array}{r} 77 \\ 34 \\ 99 \\ \hline 210 \end{array}$$

Doctor: "That's right. Let me see you write 'Martin'."
Patient: (Writes "Martin").
Doctor: "Name each letter."
Patient: "M-a-r-t-i-m" (correct to "n").
Doctor: "Why did you put 'm,' for fun?"
Patient: "I guess so."

In this visit he did a more complex and difficult problem than he had done before—he could "go me one better." That the former, or original, numbers were in his mind was perhaps suggested by the presence of much the same figures: 7, 3 and 9. Then, when he wrote the name he did so correctly till the last letter; with that he went too far and added a third loop. Perhaps there was an element of reversal in this, or perhaps it was still perversity. As is shown in his visit of a year and a half later (Feb. 8, 1936), he had reason for disliking the name "Martin."

When the patient came again, four months later, he brought a report of satisfactory progress and deportment in school. In this visit the word "Martin" was again brought up. At first he spelled it "M-a-r-t-o-n." I wrote this on the blackboard, and then he misspelled the word ten ways, I writing each as he gave it until we had the eleven misspelled attempts all before him.

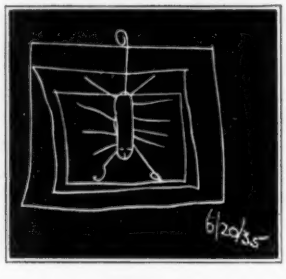

Fig. 1.—Spider-like bug in a web.

A month later, after spelling "Martin" "M-a-r-t-n," "M-a-r-t-u-n," "M-a-r-t-a-n" and "M-a-r-t-i-o-n," he said, with a laugh: "I have funny ideas. I'm stubborn."

Two weeks later I asked him again about the word "Martin." He said: "Tell me the name, the first name." I wrote the alphabet on the board and added to each letter a name beginning with that letter "M" the first letter of "Martin." I finished the alphabet. He watched me but made no comment. Then I asked him to name the words; when he came to "Martin" he recognized it, but in spelling it, even when looking directly at it, he spelled it "M-a-r-t-n-i" and then immediately corrected himself. I asked for his dreams, and he told me of being frightened at what he had seen and drew a picture of a "bug." It was a diagrammatic spider-web, with a long, slender-bodied spider in the center. When I suggested that some one might eat the "bug," he became frightened and finally said that it might grow up to be a big man and eat him up, after putting him in a ransom cave. He went into a panic, hid his head and refused to look at the board until I erased the picture. He then told me that he had seen a bug in his room and had been terrified until his mother had killed it.

The misspelling of the last two letters of "Martin" may have been reversal, but it was just the syllable that he had always misspelled; so it seemed more like

conscious avoidance than unconscious reversal. It is to be remembered that this was only a few weeks after he had misspelled the word ten times. When I wrote the alphabet and added a name to each letter, he showed no sign of recognition when I wrote the name "Martin."

The bug was like a spider; its shape was strongly phallic. A few months later another boy drew a web with a spider in the center; the body of the spider was a human head and face. A small human figure was caught in the web. The conversation clearly showed me to be the spider and the patient my victim. Adults trap the young and destroy their confidence. I was trapping him with my persistent attention to the word. While he was demanding that I erase the picture, he bit my finger. That was just as I had asked him if somebody was going to eat the bug.

In the next session I told him that he knew a great deal more than he pretended to know, and I again asked him to write the word. He wrote "clase," saying "Charles," as he did so. The "a" could easily be called an "o." and when

Fig. 2.—The writing was already on the board. The drawing was added by the patient whose case is reported in this paper. The first three words were innocuous and are omitted. The boy preceding the patient had misread the first of the bad words as "shoe," which I had written as shown in the illustration, without comment from either of us. For "p" he had read "plutz"; for "bathroom" he first read "brother" and then "bad boy" and then had correctly read "toilet" as I wrote it. "F" he read as "cock."

I said that he had come "close" many times he grinned his appreciation. When I asked him what the word was, he immediately said "Martin" but misspelled it "M-a-r-t-i-o-n."

When he came again, I had left a list of words on the board, in which list there were the three commonest short offensive Anglo-Saxon words, beginning with "s," "p" and "f." The boy for whom I had written this list had written the word "cock" beside the word beginning with "f." When this patient came in he glanced at the list, then took a piece of chalk and drew the profile of a house with a phallic chimney beside the word "cock" and, just underneath, a small Indian tent. When asked what the tent was, he so obviously dodged the word "teepee" that it was unmistakable that he was dodging, and he ended with saying: "I don't know how to say it." It was apparent that he was face to face with the threat of dangerous words. The shape of the chimney was unmistakable, but his

face and conduct were noncommittal. There was no suggestion that he could read any of the words, and when, finally, by our combined efforts, he was led to call it a "teepee "he said: "I never know they call it that." The use of the present tense suggests it was something he carried around with him for use when he needed it.

At the next visit I spelled "Martin" for him and asked him if he spelled it wrong on purpose. He replied: "If I did something wrong I'd be bawling." When I asked if he remembered any words that were on the board, he said: "Yes, 'son-of-a-bitch'." When I asked him if there was anything else, he said: "Cockroach," and when I said that there were worse words than that, he answered: "I don't want to say them" and then told that his sister and he yelled too much and had received a "licking." This ended with a dream of his stepfather and punishment. His attitude during his exposure to this list was a perfect portrayal of innocence, except for the drawings and his avoidance of the word "teepee."

Two weeks later he told of wetting his bed and his pants after his mother had spanked him. When I asked him if he was afraid that he would lose his penis if he misbehaved with it, he said "Yes" and tried to bite my finger. This led to the following conversation:

Doctor: "You want to bite my thing off?"

Patient: "Yes."

Doctor: "Because you think then yours will be bigger?"

Patient: "Yes."

Doctor: "You'd be like a girl if they were cut off, would you?"

At this point he broke out with indignation and rapidly told how his stepfather "and a lot of guys tried to take my clothes off. Opened the 'barn door' (a childhood term for the fly) to tease me. Ever since then I've been scared."

Doctor: "When they started to open the 'barn door,' you thought they were going to cut it off?"

Patient: "They started to pinch it."

Doctor: "Were you wetting your bed?"

Patient: "No, I started wetting my pants when I woke up."

Two weeks later he spelled "Martin" "M-a-r-t-i-o-n," and "M-a-r-t-e-o-n" and said: "I just don't want to spell it," but as I started to write it, he promptly spelled it correctly and then wrote it with a lead-pencil on the painted brick wall of the room, but so small that the word was only about an inch (2.5 cm.) long.

When he came a month later, I asked him the word we were trying to spell. He replied: "That was goofy" and promptly went where he had written the word and spelled it correctly. There was no suggestion of reversal at this time.

Two months later, when he was seen again, I asked him what the word was and said, as he hesitated: "It begins with M"; he replied: "Martin" and spelled it correctly. But when he started to write it he seemed confused, spelled it aloud wrong several times but finally correctly and wrote it correctly. I reminded him that there might be many words that Al (his stepfather) had used and said: "You don't like to say them except when you get mad." He replied: "It goes out of my mouth before I can stop it." He added: "I get mad at mother and I tell her to go . . ."; then he stopped, and to my remark: "What did you say?" he replied: "Son-of-a-bitch." Later in the session he again expressed anger at his mother and told again of his stepfather and his friends and their threatened attack on him, which led to the following conversation:

Doctor: "What did they say?"

Patient: "Nasty words. 'Let's get his leaker.'"

Doctor: "Is that what they really called it?"
Patient: "Called it balls."

Later, with his arm around my shoulder, I said: "What was the word we used to spell?" He promptly answered "Martin" and spelled it correctly. When I asked him to write it he wrote "Nartin," then returned and added the first loop to make an "M."

Doctor: "How did you use to spell it?"
Patient: (Spelled it in several different ways.)
Doctor: "Why was that?"
Patient: "Just to put it down."
Doctor: "Just to fool me."

He laughed and hid his face on my shoulder. I said: "Don't you realize I was a little boy once, and I know all those dirty words? Aren't there words you know? Will you write one?" He answered: "No. That's worse." To my "Ah, g'wan!" he suddenly ejaculated the excretory word "s——" and then said he did not like "kids" at his house because "they say dirty words." To my question: "What does Martin have to do with 's——'?" he replied: "He threw a rock at me. Said he'd knock the s—— out of me." "A boy whose name was like Martin?" "Yes, you dirty son-of-a-bitch!"

Doctor: "When was that?"
Patient: "A long time ago."
Doctor: What was his name?"
Patient: (Takes a long breath and refuses to say.)
Doctor: "Are you afraid to say it?"
Patient: "Yes, his name's Buddy."
Doctor: "Like Martin?"
Patient: "Martin. I used to run every time I saw him."

He then went on to tell of his stepfather threatening him for swearing and swearing himself. When I said: "I'm glad you told me about Martin. I always wanted to know why you couldn't spell Martin," he again spelled it correctly and quickly. From all I could learn, Buddy Martin, whom he later said the "kids" called "Bloody Martin," had "lain for him after school," shortly before we first had the spelling of "Martin" as a test of his abilities. He feared Martin and he feared Al (his stepfather), and when he feared he hated. As with the picture of the "bug," he was frightened by what he saw. If the word was spelled in an unmistakable way it was to be feared. If the word was exact there was no dodging it. Also, to misspell it was to show it a certain amount of contempt. It was as though it was not to be recognized (like Soviet Russia). The experience of the threatened castration was again brought up. While we talked of it we played "catch," and he threw the ball harder and harder. Then, when the dirty words had been said and recognized he told of his experience with Martin.

In the next visit he went back to the experience with Martin, described how Martin lay in wait for him after school, spelled the name correctly and then mentioned writing it in different ways. I asked: "Why do you spell 'Martin' wrong in every way you can think of?" He replied: "Being funny."

Doctor: "Well, you were mad, and I'm big like your stepfather."
Patient: "I don't like him, anyway, because I'm scared."

"Being funny" in childhood is taking a malicious delight in fooling some one. To fool a grown man had become one of his important duties. He feared Al but must not let it be seen how much he hated him.

In the next visit, ten days later, he promptly spelled the word correctly when I said, pointing to the blackboard: "What word shall we write?" and wrote it correctly.

Twelve days later he was quiet and sullen. I had left the word "taecher" on the board. He glanced at it, said it was wrong and rubbed out the "a" and "e" and put them in their proper sequence. Then he told of stealing a pin from his mother's drawer and selling it for 30 cents, and he cried bitterly. When he mentioned his fear of having to go to "that place" or when I tried to get him to tell me what place he meant, he was quite unable to remember the word "jail." Because I did not act surprised and upbraid him as he told of his crime, he thought that I already knew about it and said in an accusatory tone: "She told you, too," as though his mother was ready to take the side of the world against him.

A week later he wrote 134 and under it the correct answer. Then I wrote

$$\times 6$$

"doctor," "lawyer," "merchant," "chief," one word under the other. As he was pronouncing the first word clearly, I was writing "merchant." "Lawyer" he called "waller," hesitated over "merchant," spelled it and said "mishman." Then,

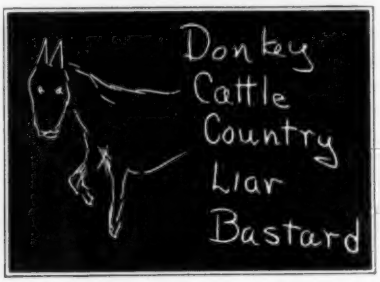

Fig. 3.—The words "donkey," etc., were written first; then, when the boy refused to pronounce "donkey," I drew the figure.

as he read "chief" and I started the next word, he chanted the rest of the list. When I asked him to repeat the list, he did so but called "lawyer" "waller" and "merchant" "mishman," as in the first attempt. This is a list of words children delight in rattling off. This is play with words. In such play children make all sorts of variations. The last time he had been confronted by me with a list of words, there were dangerous words, and he had become mixed up with them. I was the man who mixed him up. Doctor was the mix-man, not far from "mishman." He must have read the words for what they were rather than for what he miscalled them. After he had read inwardly "doctor," "lawyer," "merchant," "chief" there was nothing surprising in his going on. But "doctor," "waller," "mishman," "chief" do not strongly suggest the rest of the list. Having in mind the boy's stubbornness, I wrote a second list of words, beginning with "donkey," a word that combines the intimation of stubbornness and foolishness. He spelled it correctly. As he finished spelling it, which he did slowly, I had finished writing the list of five words.

He pronounced the first word "donker." Then, he spelled it "d-o-n-k-r-y," adding: "I can't read." After I pointed to the rest of the words, I asked: "Is there any word you know?" He said: "A mule or a horse." I then drew the figure to the left of the list, whereon he promptly said "donkey" and I laughingly

said: "If you could see the way you batted your eyes when you looked at the picture!" He laughingly replied: "I knew it all the time." He had glanced down the list as I wrote it, and as he was attempting to pronounce the first word, the "r" sound, important in the endings of the last three words, was used. It had apparently stuck in his mind.

Then he spelled "c-a-t-t-l-e" but made no effort to pronounce it; so I put horns on the animal. There was no response. To suggest the sound, I wrote the first syllable. This he frankly resisted by pronouncing the "a" as in "Kate." Of course he knew "cat." To my "What?" he gave in and said: "Cat." But to my question: "What has horns?" he answered: "A cat has"; then to my indignant "Is that so?" he kept up the dodge, saying "cows" instead of "cattle." He further responded to my question: "What do you call a lot of cows?" with "cat . . . cat." The conversation went on:

Doctor: "When you look right at it you won't call it 'cattle,' will you? What's the next word?"

Patient: "C-o-u-n-t-r-y" (in spelling, stammers on the "c" sound).

Doctor: (after several seconds) "Where do cows run?"

Patient: "Countries."

Doctor: "What's the next word?"

Patient: "Layer."

Doctor: "What do you call me?"

Patient: "Liar."

Doctor: "What's next?"

Patient: "B-a-s-t-a-r" (spelling).

Doctor: "Why don't you give it a 'd'?"

Patient: "Bastar (ignoring the "d" sound).

If he was lying to me, if he knew the words but chose to cover it up or if the ruling interest in his mind was to cover up his knowledge rather than to read correctly, the word "liar" could stand there as a threat, as well as "bastard." There was, of course, an element of threat of the same sort in the sound of the word "donkey" when applied to a stubborn child who was refusing to go along with me because he would not rather than because he could not.

"Bastard" he refused to complete, even after I called his attention to his failure. It was a word he did not like; he would not give it the final "d." The addition of that letter makes another sound definite, a word common to boyhood but taboo to usage between children and adults: the excretory or obscene word "terd" as applied to feces. I then wrote "luck," "pluck" and "duck." He read "luck" with no hesitation and spelled "p-l-u-c-k," without saying the word. He spelled "duck" but again would not take the risk of pronunciation. He lay down as though tired. It was my conviction that the word "f——" was in his mind and that he would not take the chance of saying a word so close to it, for fear he might give voice to his thoughts. Therefore I wrote a synonym, also a vulgarity, "screw." This he misspelled "s-c-r-e-y" and added "sacred." He called "w" "y," a letter near "w." There was obviously no reversal in this. He did not call "w" "u" as he might have if reversal or upside down or mere physical appearance were the only mechanism at work. When he named the word, one might say he went as far from it as possible—from the lewd, profane and vulgar to "sacred." Then he dodged recognition as long as possible, calling a picture of a screw "ice-cream cone," "knife," "sword," "spear" and "nail". We were in the very middle of the forbidden words. If he showed that he recognized these words, an adult would know that he had guilty knowledge, knowledge he should not have, etc. I finally pretended to be using a screw-driver on a screw in the

window handle, and he had to say the word "screw." Then he tried to hit me and throw me to the floor. When I asked: "What other word ought to go with 'luck' and 'duck'?" his lips started to form an "f," but he refused to go through with the word and fell back on the semblance and expression of ignorance with "I don't know."

Doctor: "Screw."

Patient: "You said it."

In the next visit I called attention to his trousers being partly unbuttoned in front and spoke of Al. When I said, with a smile: "A screw," he drew a screw. It was as though we were going on with our conversation of the last visit, four days before. This visit exemplified the influence of the human body in the establishing of symbols. Beside the screw he drew a nut, which he later embellished, as seen in the picture. Nuts—meat inside a shell, also a machine nut. He said: "Screw balls are in the bag" (bag is a boyhood term for scrotum), and: "The bag is by the bat." "Bat" then led to a drawing, a diagrammatic or symbolic figure of male genitals, in the right lower part of the board, at which

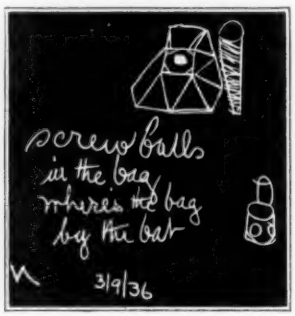

Fig. 4.—The figure in the upper right corner is that of a screw; beside it is drawn a nut, with disguising embellishments. "Screw balls" and so forth were written as the patient said them. The other figure is a diagrammatic drawing of the genitals.

he made a "p-s-st" sound and then said: "Could leak as high as that," pointing half way up the wall. There was freeedom in his way of talking never before shown.

A week later he returned. On these last visits he came by himself. It was bitterly cold, and I cannot but think that he came because he wanted to. He lived in another part of the city and had to change cars twice on the trip. He was not warmly dressed—no overcoat, no mittens.

In this session I asked him to read a paragraph in a magazine, beginning: "I know." This he repeatedly read: "I knew." I called his attention to this and, a little later, said: "You are smarter than you let any one guess." He muttered: "I know something." Then he told a story of guilty knowledge. "Nobody knows how I found that out" began the account, and then, in disjointed, halting phrases: "I saw what it was. Not having children, not wanting them. A sign in a store window, upside down. I couldn't read it. My boy friend did. Boys shouldn't know it . . . where ladies get their babies. I asked what it was. We walked down the street. He said: 'Come on. Let's go to the candy store.' I said: 'Here's the candy store,' but he never went in. He said: 'Wait

a minute, and I'll tell you." (Pause) "Now I've broke my promise." (This last was wailed.) After I had reassured him, he added: "I told you we couldn't read it 'cause the sign was upside down." (Pause) "He told me we came from Mama. But (in a most apprehensive tone) "don't tell her."

He knew what adults pretend children should not know. This need to pretend ignorance so that he may be thought to be innocent may play an important part in all the so-called stupidity of the mentally retarded child. To cover up the possession of such guilty knowledge or evil thoughts, "I don't know" is the simplest, as well as the most useful, expression and attitude. Children say "I don't know" so easily—"I don't know," said with an open, scarcely moving mouth, all in one sound. If it is adhered to there is nothing the potential threat, the attendant adult, can do. It is of the nature of forbidden knowledge, or, in Biblical terms, it is the knowledge of good and evil. When the boy attempted to tell what it was that he should not know he avoided it. He told how his friend tried to avoid saying it—"Let's go to the candy store." Of course, this was merely a repetition of the adult attitude: "I've told you, but you mustn't tell any one. Those aren't things to talk about with other children"—all the ways of making the possession of such knowledge a secret or shameful thing.

There were a number of reasons for this boy's wanting to appear innocent (ignorant). Perhaps the most important obvious reasons were connected with the figure of his stepfather, Al. Al was a "tough guy," a veteran. He drank, and when he and his friends were drinking they knew how to scare the boy. This was by a threat of an attack on his genitals. An attack of this sort is obviously of the nature of punishment. The boy's hatred of Al made him that much more vulnerable—the more he hated the more he must fear an attack. Al had shown him two forms of punishment: the strap (a "licking") and "pinching it off." The latter was in the realm of forbidden knowledge, of the unmentionable. There was no suggestion at any time of reprehensible experiences of a sexual nature. There were, however, plenty of instances of the possession of forbidden knowledge. It was obvious in the last session reported. He told the story disjointedly, said he had broken his promise, before he had done more than hint what it was all about, intimated that the sign in the drug store had something to do with contraception and then finally said: "He told me that we came from Mama." Right up to the end were expressions of stupidity, mental inadequacy and the like. "I told you I can't remember," "I don't know," etc. Of course, it is quite possible that this, again, was only a partial revelation. However, it was a partial answer to that all-important question of childhood: "Where did I come from, and how was I made?" It was some knowledge of the workings of the human body. In his attitude toward the list of words that led to the drawing of the phallic chimney and the teepee, there was no suggestion that he knew them. However, he drew the house with its phallic chimney full up against the appropriate word and then added the teepee. Many children have drawn houses for me, and almost all of them have chimneys. In fact, not infrequently, as they add the chimney with the smoke blowing, some remark will be made to go with it. However, no child has ever drawn such a definitely phallic chimney as this. In fact, it is almost always square or rectangular. Also, when the patient returned two weeks later and I asked him what words were on the board, he replied: "Son-of-a-bitch." "What else?" "Cockroach." Failure of recognition of the import of the words was further attempted in his strict avoidance of "teepee." He would draw it, but he must not know or say the word. He would not read the word when it was written out for him.

In the session, a week later, he showed a swollen, infected finger. This I associated with the idea that "dirty" thoughts cause an erection. I then drew the house with the phallic chimney and the teepee. The latter he called: "Indian wigwam. No (as I showed I was waiting), wigloo." When I wrote "tee" he spelled it as though he had never seen it before, "t-e-e," and when I said: "What else?" he grinned and said: "Teepee." "Why do you grin like that? Is 'teepee' like 'pee'?" He answered with: "It ain't; it ain't got no leaves." Obviously, this referred to tea-leaves and showed a facility of association of words far from inadequate. I then wrote a list identical with the one that had evolved the phallic chimney and teepee. He read the first three quite readily; they were innocuous. But the fourth, a taboo word, "s——," he promptly read "shut." It was followed in the list by the word "cover," and as he saw I did not accept "shut," he corrected it with "sit." The influence of "cover" must have helped to determine the choice of "shut" and "sit." He was looking ahead and the ideas suggested by "cover" gave direction to the avoidance necessary to escape the dangerous word. The last word I had not written, but as I started with "fu,"

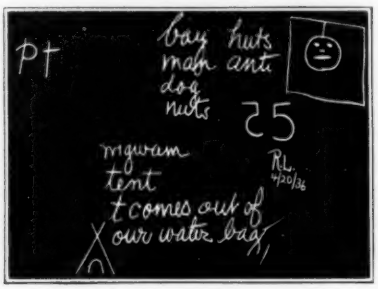

Fig. 5.—The patient erased the figure in the upper right corner but left enough for me to line it in again. I failed to draw more than the outline of the web. I drew the two "5's" to show how he had started the web and the circumference of the head. This seems like plain reversal.

he pronounced the letters "f-u" and then said as quickly as he could: "Buck." Then he asked to go to the toilet, saying as he went out the door: "You're safe if your mother don't catch you."

A week later he spoke of his teacher in mocking tones: "Miss Ough" (as one expresses pain) . . . "bright," he called her. I remarked that it hurt to be smart and said: "There used to be a word . . ." He promptly spelled "M-a-r-t-i-o-n," and when I said, as though astonished: "What?" he immediately said: "i-n." "Why did you say 'i-o-n'?" was answered with: "To see if you'd notice it." "When you said it, that was just as though you didn't know it. I suppose that's what you do with Teacher" evoked: "Sure, when I want to."

I again drew the teepee, saying: "What was it?" "Wigwam; wigaboo," he said. He wrote a large "T" (in the upper left corner), saying: Comes out of our water bag." I put a "P" before his "T," saying: "Now will you say it?" When there was no response, I said: Suppose you turn it around," and he answered: "Teepee." To my: "What's that?" he said: "Tent." "Why wouldn't you say it?" brought: "It's always that."

Doctor: "I'll write some words, and you tell me what they are." (I wrote the list: "boy," "man," "dog," "nuts," "huts" and "ants.")

The first word he called "bag." I said: "What's in the water bag?" He said: "All my guts." He then drew two 5's. One faced correctly; the other was written in reverse and completed a frame, the top of which began with the horizontal stroke at the top of the 5. (I drew the two 5's in the picture.) This evolved finally into a spider-web and was so named by him. As he drew the face, I said: "What kind of a guy is that?"

Patient: "Nigger; nuts."

Doctor: "Finally you said it, didn't you?"

Patient: "I didn't say anything."

And then I said again: "Of course, kids think adults try to catch them" (the spider and his web). He came back to the guilty knowledge and told of the sign. It was upside down: the "insides" (the fetus).

At the next session, eight days later, he came in singing "Three Blind Mice." When I suggested that he was afraid of me, as the blind mice would have been, he became angry and said: "Nuts to that! Rats to that!" (This shows the tendency to play with words as well as the other associations.)

He then told the story of a boy who had an infected tooth and would have to have it drilled and "all the dirt cleared up." I likened "dirty" words to dirt in the mouth, and he said that he was glad that "he was not that kid."

Doctor: "All kids like to put things in their mouths."

Patient: "Not these (pointing to his genitals); I couldn't get them up there."

Doctor: "How do things get inside of people?"

Patient: "By eating."

Here he called attention to a bird 40 feet (12 meters) away, outside the window, saying that it was a squirrel. This he repeated two or three times. (This took place in the city, where there are no squirrels.)

Doctor: "A squirrel gets nuts, and if a lady swallowed a nut . . . ?"

Patient: "That's a seed."

Doctor: "You saw a sign of a baby inside mother, upside down."

Patient: "I didn't know how."

Doctor: "You kids like to know things."

Then he told the story of the three boys and their mother and father. The first two boys were "dumb." The third studied. A tree had to be cut down. The two older ones failed because they did not learn to make the ax say "chop, chop." The youngest learned just the right words to use and cut down the tree, and "he freed his brother, and the king died, and he grew up to be a very fine man and married the queen." If the ax did not say just the right thing, each time that a limb was cut off it grew back again twice as big.

I then asked him if he was afraid he might know something; he replied: "You promise not to snitch on me," but before he would tell me anything, he played with words, saying: "Eeny, meeny, miney, mo," and then passed wind and called it a stink bomb. When I wrote four words on the board, the last one the boyhood word for breaking wind "f——," he read the words as quickly as I wrote them and added: "I didn't want to show I knew it, just to see what you would do. I knew it all the time." I then told him how babies grew and were carried in the uterus. He asked "if the lady had to have an operation"; so I described labor. He said he feared to know about babies; his mother would bawl him out and give him a spanking. I then wrote a list of seven words containing the "f——" word for intercourse. He pronounced the first word "house" "house," then giggled and tried to erase the words. He acted so angry that I erased the words, saying I was sorry I did not let him erase them. He then wrote the word "f——" quite well, saying: "I knew it all the time." He told of the "worst thing" he ever

did, soiling himself in school about four years before. Then he said: "Horses and dogs have to go to the bathroom to do number two" and added: "If horse s—— is put on grass it helps seeds grow—grows green." Then: "I thought of something. You can't see it with a microscope; when a baby gets a little older and its bowels get to working, it all comes out of its mother," thus showing he must have known about pregnancy and thought about the baby's excretion before I said anything about it. In saying of horses and dogs that "they go to the bathroom to do number two," he minced his words in an affected manner and then lapsed into the vernacular. However, he avoided putting seeds and excreta together but spoke of an ordinary and acceptable process of fertilization. This led to the question in his mind of how the supposed fetal excreta would be taken care of.

In the next session, after friendly play, he corrected me in a mistake, and in answer to my question: "I'm trying to understand whether the sign scared you so you couldn't read, or was it the other kid?" he said: "I don't like reading 'cause it's none of my affair, none of my business. I'm afraid I'll read the wrong thing and get bawled out for it, bawled out proper, I mean." He then told of being called "dummy" and "puss face." When I asked him to write "puss face," he wrote "bill." The "b" was first written like an "f"; then the lower loop was

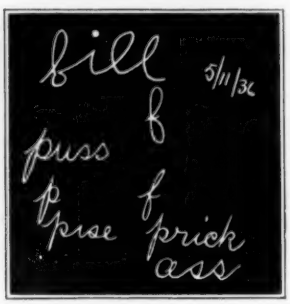

Fig. 6.—The "b" of "bill" shows the short lower loop which was added as the patient erased the longer loop of an "f."

erased and shortened to make a "b." The word is innocuous, but if he had left the "f" and followed with the "u" of "puss," he would have been on the way to a poisonous word. Also, if the original "f" had been a "p," a sound similar to "b," the "pi" was on the way to another poisonous word. Either of these words applied to his face would be intolerable, but because of his way of writing "bill" for "puss," I said: "I'd write 'puss' this way—'puss.' But if it was "i" instead of 'u'?" "Pice" ("i" sounded as in ice), he said. To my remark: "You made the 'b' like an 'f,' he said: "I thought you'd get all mixed up"; then, "I'm a dumb-bell."

COMMENT

In the course of the development of the human personality there are many ways by which a sense of superiority can be established and maintained. In general, the fear of being and doing wrong outweighs the sense of security or the feeling of being right. In left handedness there is the sense of being wrong. Through a combination of circumstances and coincidences, the fear of being wrong may become a potent factor in preventing the acquisition of language. Since

words play such an all-important rôle in academic learning, fear of words may seriously prevent progress in such learning. Such fear may constitute an emotional barrier to reading. There is an element of adventure in reading for the young. The young may look on the printed page with apprehension. There is always the unknown in the words ahead. Many children look ahead as though to guard against the possible trap or danger. "Better be safe than sorry."

Like many other children, the boy whose case has been described showed marked differences in responsiveness when he was tested or questioned and when he was free from pressure. One was forced to assume that resentment was aroused as soon as learning was required. In analytic work resentment is so often seen to be the outcome of fear that such an origin was thought possible in this case. That this was so is shown in many places in the record. He feared and resented his teacher as he feared and resented his stepfather. He feared and hated a boy named Martin; he refused to spell the name correctly for a year and a half. In his unwillingness to spell the name correctly he showed ingenuity quite out of line with a conception of dulness.

He frequently showed unwillingness to display knowledge, especially of words. This unwillingness was apparently due to a fear lest possession of knowledge should lead to punishment. Therefore, to say "I don't know" and to cultivate and assume an appearance of ignorance were desirable. On the other hand, there were an alert responsiveness and a desire to know about "where he came from." From the general attitude, as well as from the assertions of this boy, it was easy to be convinced that he did not know many of the things he did know. If such an appearance of ignorance and degree of retardation could be assumed at 10 or 12 years of age, it was to be expected that they need not be assumed much longer: They would become "natural" or real.

CONCLUSIONS

The fear of words can lead to evasions that interfere with learning to read. Looking ahead to avoid trouble may lead to difficulties with recognition and pronunciation of the word at hand. This may have some relationship to such mechanisms as reversal. Children, not knowing words, can be led into fears of meeting taboo words. Such fears lead to resentments and rejections of learning. Most taboo words have to do with natural processes of the body. Desire to know about these processes is inevitable. Expressions of that desire are frowned on. Words are the natural means of expression. Knowledge is feared and resented.

The impropriety and fallacy of generalizing on the basis of one case have often been stressed. It is impossible to work out a large

number of cases in great detail. The idea that emotions interfere with learning was suggested many years ago by a series of children with some sign of physical damage to the brain who were fairly adequate until school age and then, when they were thrown in competition with their equals (in age), physical signs of fear developed, and therewith they seemed to show an increasing amount of retardation.

Recently, a teacher verified my findings in a boy whose history will be published in more detail later. He was 9 years of age, was left handed, had been in the first grade for two years and had spent two years in an orphanage. He told me on his first visit that he did not play with boys "because they swear." In the second visit he spoke of his little dog and said that it "spit on the snow." At the next visit, after writing his first name readily, he volunteered: "I can't write my last name" (it began with "P"). When asked for the alphabet he said "a-b-c" and then wrote "p" for "d." When asked what letter followed "c" he answered: " I don't know," then; "a-b-c-, tumbled-down d. The cat's in the fiddle and can't see me." He then said he hated school—"too many teachers holler at me." Later, when asked whether he was scared of what he might learn, he answered that he might learn "bad stories" and "bad words" and that his mother knocked his tooth out when he said that the dog did not p—— on the floor.

Later, he boasted: "I don't know how to write "boy." You could take some letter off and make dog." He then juggled letters, bringing in the letter "p" where it did not belong. Later, in answer to the question: "Did you ever learn anything you wished you didn't know?" he answered: "Just words." This reaction occurred on a day when I had put a list of ten words on the board, three of them being the short and dirty words for excreta and intercourse. In his reaction to these words dodging was obvious, although the pretense of ignorance was convincing.

Also, as he started to read the first word, "mother," I could see his eyes fall to the first offensive word, the fourth in the list. He read "mother" as "mama," and then misspelled the next word, "train," "t-r-o-i-a-i-n." misspelled the next, "dirt," "t-i-r-t," pronounced it "dog," and the like. Throughout this halting attempt I could see his eyes travel up and down the vertical list. It was obvious that he was distracted from the word he was attempting to read by thoughts suggested by the words he knew he was going to meet.

Later, I wrote another list of words, without any bad ones. There was the same attempt to fool me, and, more important, the tendency to look ahead in the list while attempting to pronounce a word was obvious; in pronunciation he mixed up letters or sounds that suggested dangerous ideas.

The rest of the history bears out the impression already indicated. The teacher reported that the boy had been in her special room for about a year and a half, during which time she had cultivated and apparently gained his confidence. He seemed quite unable to read. Finally, to her complete surprise, he announced to her before the entire class that he knew why he could not read: He was afraid of the dirty words that were in the book they were working on. In support of this statement, against her surprised denial of the presence of offensive words, he told her that his older brother was helping him in home work in reading and that he (to quote the teacher's report) "supplied the missing words, and they were always very dirty ones." The boy would not say them aloud but whispered them in the teacher's ear. One can see the picture of the older brother ejaculating his exasperation. "From that time (the patient) started to read, and, although his progress has been slow (the teacher thinks) that ultimately he will be a normal reader."

THE KLIPPEL-FEIL SYNDROME

A PATHOLOGIC REPORT

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Hitherto, reports of cases of the Klippel-Feil syndrome have considered only the clinical manifestations. These have been described as shortening of the neck, low hair line, limitation of movements of the head, abnormal associated or mirror movements, clumsiness of the hands, disturbances of deep sensation in the hands and forms of ocular palsy, affecting chiefly the third cranial nerve. Roger, Arnaud and Audier [1] discussed the nervous manifestations associated with the Klippel-Feil syndrome, reporting an instance of their own and reviewing in detail the cases reported up to 1934.

The following case is described, because pathologic reports on the changes in the spinal cord are lacking.

REPORT OF A CASE

History.—A deformed, full term girl (fig. 1) was delivered from a primipara aged 20, who had had a profuse vaginal discharge during the entire pregnancy and toxemia of nephritic origin in the last trimester. The general appearance of the infant was that of an old person. The head was in extension, and the hair line was low, being on the upper part of the back. Torticollis was present to the left. The palpebral fissures were wide, and there was left internal strabismus. The scapulae were located high on the thorax, as is characteristic of Sprengel's deformity. The upper extremities were in flexion, with spasticity of the fingers and hands. The carpal bones appeared larger than normal. Mirror movements of the hands and arms were apparent. The lower extremities were abnormally long in relation to the trunk. The formation of the feet was normal. The child was able to suckle but had great difficulty in swallowing. Death occurred on the thirty-fifth day, from malnutrition.

Gross Pathologic Observations.—Postmortem examination by Dr. J. J. Moore, pathologist to the Ravenswood Hospital, revealed a marantic, emaciated infant, with wrinkled skin and the facial expression of an old person. The body weighed 4¾ pounds (2,154 Gm.). and was 46 cm. long. The face was triangular, with a broad forehead formed by large oval frontal bosses and a narrow, pointed chin. The head rested directly on the thorax. There was marked torticollis to the left. The head could be rotated only through 20 degrees; it could not be flexed on the chest and could be moved only slightly backward. The greatest transverse diameter

1. Roger, H.; Arnaud, M., and Audier, M.: Les manifestations nerveuses du syndrome de Klippel-Feil, Marseille-méd. **1**:233 (Feb. 15) 1934.

of the head was 11.5 cm., and the anteroposterior diameter, 12 cm.; the greatest circumference was 35 cm. The distance from the shoulder to the parietal eminence was 10 cm. on the right and 7 cm. on the left. The fontanels were open. Examination of the thorax, abdomen and pelvis revealed nothing abnormal.

The specimens removed consisted of the brain, the posterior part of the skull and the entire spinal column, containing the spinal cord. The brain did not become sufficiently hardened in a solution of formaldehyde to permit anatomic study. The spinal column and cord were preserved in 70 per cent alcohol.

There was a cartilaginous attachment between the cervical transverse processes and the deformed occipital bone, so that the posterior wall of the spinal canal, from the occiput to the fourth dorsal vertebra, contained no bony structure. The foramen magnum was oval and extended from the left to the right obliquely, instead of sagittally. It measured 4 cm. in the anteroposterior diameter, 2 cm. in the anterior part and 1 cm. in the posterior portion. The spinous processes of the cervical and the first four dorsal vertebrae were lacking. Twelve ribs were identified. The transverse processes of the first cervical vertebra were large and appeared as cervical ribs. All the anterior and posterior spinal roots could be isolated. The sacral, lumbar and thoracic pairs emerged in their normal relation-

Fig. 1.—Photograph of the infant, 5 weeks of age, showing absence of the neck.

ships; the cervical roots emerged from the lateral portion of the broad semibony mass comprising the malformed cervical vertebrae. The dorsal portion of the sympathetic chain was present.

Cross-sections of the spinal cord were taken through representative levels and were stained with hematoxylin and eosin, iron hematoxylin and the Nissl method. Fixation in alcohol did not permit study of the fiber pathways.

Microscopic Observations.—Sections through the upper part of the cervical portion of the cord revealed two lateral parts, which were nearly divided by a deep anterior fissure (fig. 2). The pia was present over the anterior aspect; over the posterior margin no pia was seen, but for some distance to each side of the midline ependymal cells were arranged lineally. The central canal was not present, but its position was indicated by an indentation of the ependyma. The cord was round and completely surrounded by pia in the lower cervical levels (fig. 3). The pia was thickened and in places comparable in structure to the dura. Abundant, heavily staining collagen was present, at times arranged in regular bundles; in other places it formed nodules and projections outward from the pia. Along

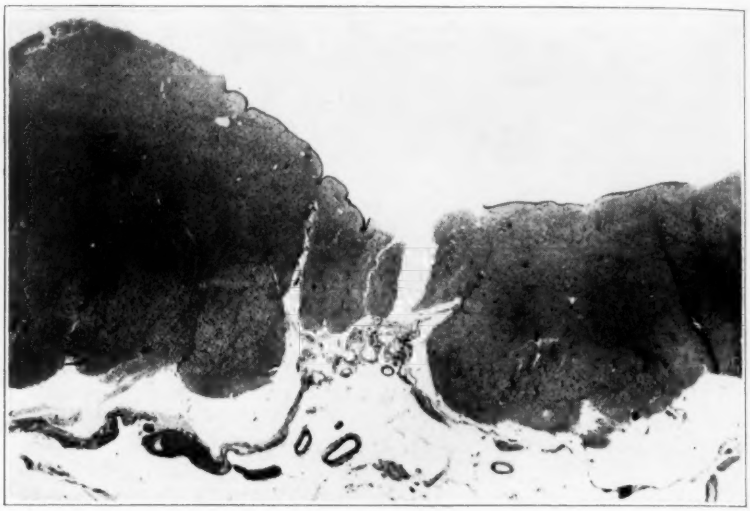

Fig. 2.—Section through an upper cervical level of the spinal cord, showing the results of incomplete fusion of the two halves of the neural plate. The posterior aspect of the cord was devoid of pia and was lined with ependymal cells.

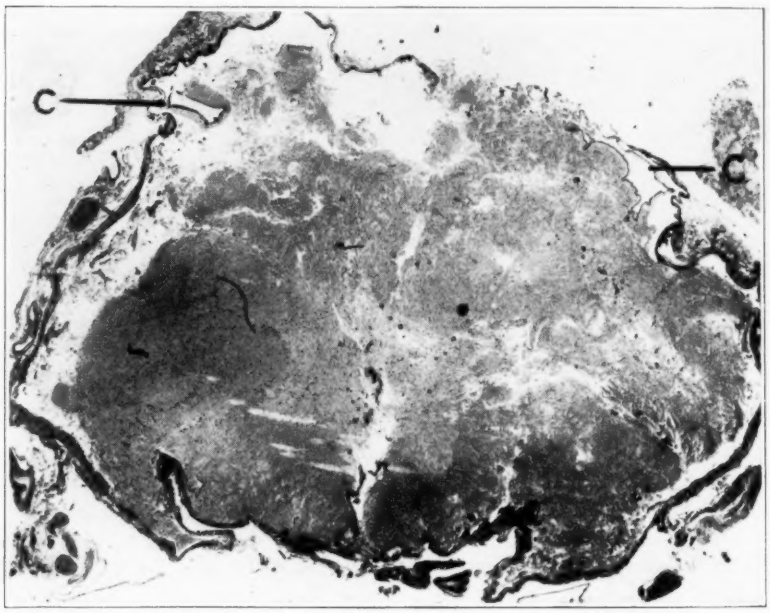

Fig. 3.—Section through a lower cervical level of the spinal cord, showing the abnormal fissures in the anterior aspect and (*C*) cavities lined with ependyma located in the posterior aspect of the cord.

the anterior aspect of the cord the pia extended into the substance of the cord, forming two abnormal fissures, one on each side of the midline. Numerous pigmented cells were seen scattered through the pia. Sections of blood vessels were numerous. In places several such sections were seen lying close together, as though a tortuous vessel had been cut. More striking was the large number of sections of vessels which appeared to consist only of smooth muscle tissue, as though the section had passed through the wall of the vessel only (fig. 4). The frequency of these vessels and the extent of the muscular tissue suggest that there had been an abnormality of vascular formation rather than that the picture depended on the plane of the section.

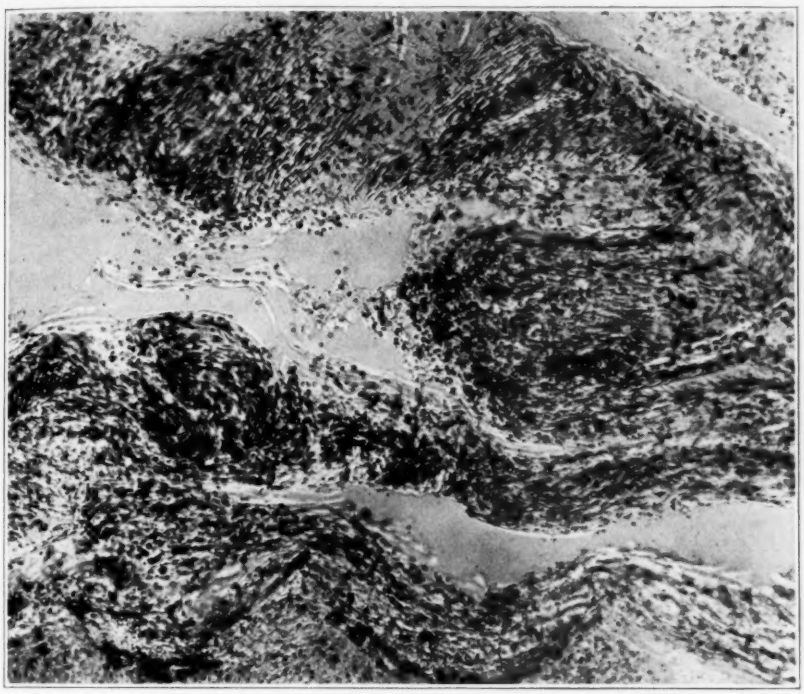

Fig. 4.—Portion of a section of the cord, showing the extensive vascular formation in the pia.

Vascular tissue was observed also in the connective tissue septums. The spinal cord at this level was completely formed. The anterior and lateral columns appeared to be normal. The central gray column could be identified. The posterior column appeared to be homogeneous, with evenly distributed glia cells and dark-staining nuclei. In places definite gliosis was present, especially in the more marginal areas. The blood vessels of the cord appeared relatively normal, except that occasionally they had thickened walls. The central canal was present, with its long axis in the anteroposterior position. It varied in size at different levels and at times appeared as a double structure, with collections of ependymal cells connecting the two cavities. A moderate degree of gliosis was present about the canal. No commissure could be identified between the two parts of the cord.

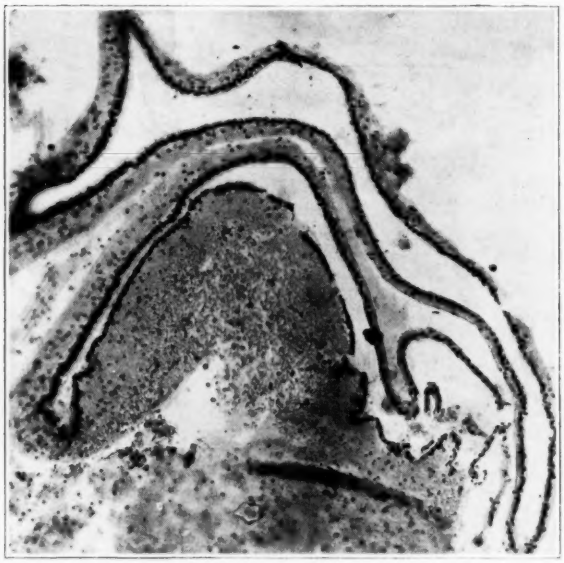

Fig. 5.—Section through the cavity on the posterior aspect of the cord in the lower cervical levels.

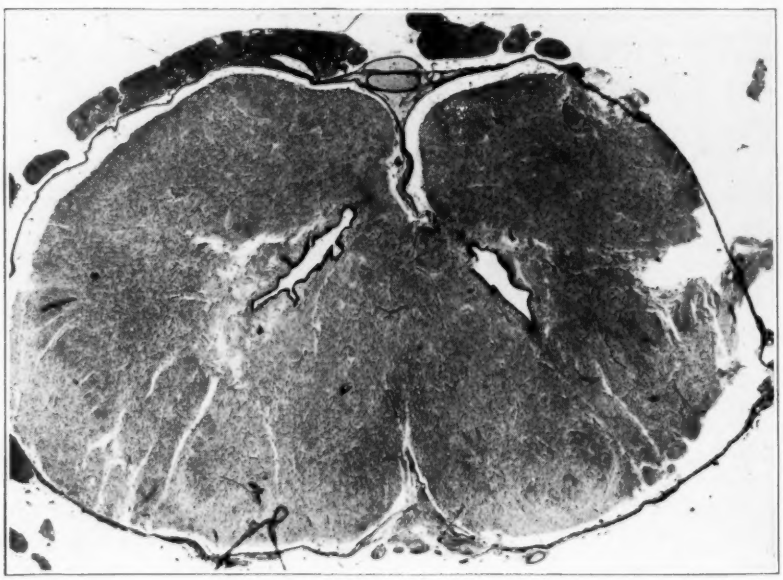

Fig. 6.—Section through the lumbar level of the spinal cord, showing the double central canal.

Ependymal cells were scattered over the posterior aspects of the cord. In some places they formed a lining to the posterior margin; in others, aggregations of cells within the substance of the posterior column or small cavities lined with ependyma. Two larger ependymal and glial structures were seen, one on each side of the midline close to the entrance of the posterior root. These cavities appeared to be partly in the posterior column and partly in the pia (fig. 5).

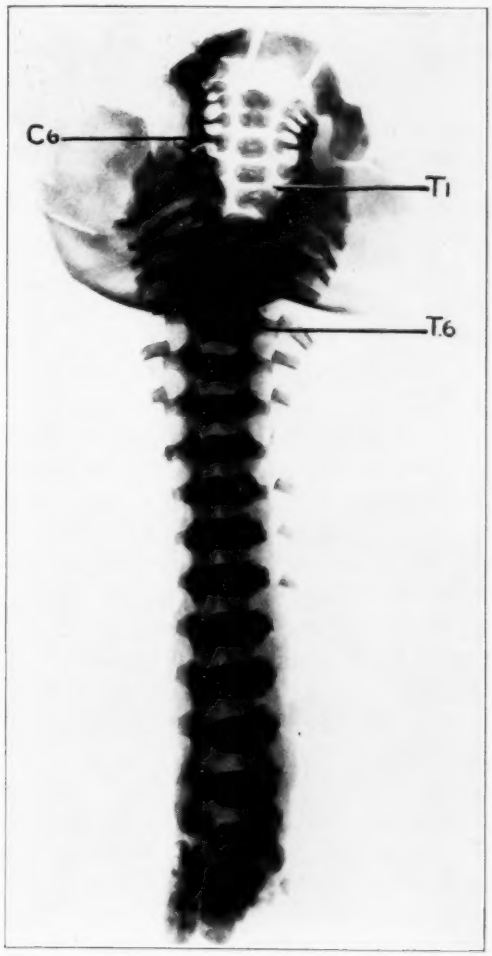

Fig. 7.—Roentgenogram of the spinal column. *T 1* indicates the body of the first thoracic vertebra; *T 6*, the body of the sixth thoracic vertebra, showing the notched character, and *C 6*, the transverse process corresponding to the body of the sixth cervical vertebra.

In the thoracic region the cord was round. In the upper levels, as in the cervical region, distinct gliosis of the posterior columns persisted. The central canal occupied the same positions as at higher levels; it was usually round but in places

elongated or double. In the lumbar region the central canal was double, being in the form of two large cavities occupying the area of the commissure and the posterior horns of the central gray column. A moderate degree of gliosis was present above the cavities (fig. 6).

Roentgenographic Findings.—A roentgenogram of the specimen consisting of the posterior part of the occipital bone and the spinal column revealed the normal number of sacral, lumbar and dorsal vertebrae (fig. 7). The second dorsal body was displaced, lying in an oblique position. The first thoracic body was partly fused to the seventh cervical vertebra, and little of the intervertebral disk was seen. The sixth cervical body appeared isolated, with wide intervertebral spaces. A mass of vertebral tissue appeared above the sixth cervical vertebra. It appeared to be composed of three fused bodies, and no intervertebral substance was seen. Six cervical transverse processes were identified on each side. The transverse processes that corresponded to the bodies of the mass consisted of four processes on each side, springing from a common base. This may indicate that more than three bodies were involved in that structure. The bodies of the vertebrae were not normal, and even as far caudad as the tenth dorsal vertebra one saw elongation of the lateral axis of the vertebral body and a small cleft in the midline. The occipital bone was definitely cleft, and the defect of the cleft was united with the defect of the cervical arches by a nonbony union.

COMMENT

The character of the defect of the bony structures merits discussion. It consisted of a cleft in the occipital bone, as well as in the arches of the first four dorsal and all the cervical vertebrae. The defect was closed by a connective tissue union between the vertebral arches and the occipital bone. This resulted in expansion of the foramen magnum, opening into a funnel-shaped canal which extended caudad as far as the fourth dorsal vertebra. This allowed for sinking of the medulla and cerebellum, such as is characteristic of the so-called malformation of Arnold and Chiari.[2] A defect of this type has been observed almost constantly in cases of the Klippel-Feil syndrome in which postmortem studies have been made. It was described by Feller and Sternberg[3] in a report of five cases of short neck in fetuses.

Since the original description of this syndrome, considerable discussion has arisen as to what is the essential defect. In the original work of Klippel and Feil it was suggested that the constituting factor was a peculiar type of occult spina bifida, but later Feil concluded that the specific defect was the numerical reduction of the vertebrae. There has been considerable opposition to the latter view, and the work of

2. Ernst, P.: Missbildungen des Zentralnervensystems, in Schwalbe, Ernst: Die Morphologie der Missbildungen des Menschen und der Tiere: Ein Hand- und Lehrbuch für Morphologen, Physiologen, praktische Aerzte und Studierende, Jena, Gustav Fischer, 1909, vol. 3.

3. Feller, A., and Sternberg, H.: Zur Kenntnis der Fehlbildungen der Wirbelsäule: IV. Die anatomischen Grundlagen des Kurzhalses (Klippel-Feilschen Syndroms), Virchows Arch. f. path. Anat. **285**:112, 1932.

Feller and Sternberg was in agreement with the original opinion of Klippel and Feil.

From a study of the vertebral bodies in the present case, it is seen that not only did abnormalities exist in the upper portion of the dorsal and in the cervical region but that throughout the dorsal vertebrae there was a cleft appearance of the bodies, seemingly indicating that the two developing halves of the body did not fuse well in the midline. What might be a hemivertebral body was seen in the instance of the second dorsal vertebra. There was also evidence that fusions occurred between the vertebral bodies. On the other hand, the segments of the vertebral column were fairly well represented by the number of vertebral arches. It is suggestive that there was numerical reduction not of the segments but only of the vertebral bodies. Also, it has been observed that in the reported cases of this syndrome in adults the vertebral reduction was more apparent than in early life. A reduction in the number of vertebral bodies, then, seems not to be primary but to have occurred through the growth of the more normally developing structures, forcing the misplaced and abnormal parts into vertebral mass formation as the body grows older, as was postulated by Müller.[4] It is surmised that the disturbances in the body formation have something to do with the precision with which the chondroplastic tissue surrounds the notochord and joins in the midline.

The character of the defect in the spinal cord was also in the nature of a cleft in the posterior aspect of the cervical and the upper portion of the dorsal region. At these levels complete formation of the spinal cord failed to occur, so that a central canal was not formed but was represented by a layer of ependymal cells lining the posterior margins of the cord. Even at lower levels, where the cord had been completely formed, the central canal was of abnormal location, size and contour. Other structures were also involved in the defect, as evidenced by gliosis of the posterior columns and distinct deviations in the character of the blood vessels and the connective tissue.

These changes are referable to defects occurring in the formation of the original neural tube and are, we believe, a true expression of dysraphia. Normally, folding of the neural plate occurs in the formation of the neural tube, with fusion of the two halves in the posterior portion of the midline. The fusion not only must be complete in the posterior part of the midline along the entire length of the tube but must occur with precision, so that there is no displacement of early, undifferentiated embryonal cells. It seems evident that if such displacement occurs tissues will be formed in abnormal locations. The defects will be also widely spread throughout the nerve structures, especially

4. Müller, Walther: Untersuchungen zur Biologie der Wirbelsäulenmissbildungen, Deutsche Ztschr. f. Chir. **242**:94, 1933.

in the posterior part of the midline, owing to the growth and expansion of the body. In this instance, while the defect in the bony canal and that in the cord were most marked in the cervical region, distinct abnormalities occurred in the formation of the central canal throughout the dorsal and lumbar regions, where no defect in the bony structures was seen. Furthermore, not only had the defect involved the nerve elements of the cord but abnormalities were present in the formation of the blood vessels and connective tissue, both in the degree of development and, in the case of the connective tissue, in position.

The importance of dysraphia as a basis for the future development of pathologic conditions of the spinal cord was first pointed out by Bielschowsky and Unger,[5] in relation to syringomyelia, and by Henneberg,[6] in relation to the formation of various types of medullary tumor. More recently, Mackay and Favill[7] described a case of combined syringomyelia and intramedullary tumor and gave a comprehensive review of the literature.

CONCLUSIONS

The Klippel-Feil syndrome may be associated with defects in the cerebrospinal axis not only in relation to the bony structures but at distant levels.

The abnormalities in the spinal cord are chiefly in relation to the nerve structures concerned with the formation of the central canal. There are also abnormalities in the development of the mesoblastic structures.

While there is a numerical reduction in the segments of the bony structures, there is no such reduction in the segments of the spinal cord.

The essential features of the Klippel-Feil syndrome are: the cleft of the occipital bone and the cervical arches, with maldevelopment of the vertebral bodies, and reduction of the vertebral bodies. The reduction in the number of vertebrae is a secondary process. In this we are in agreement with the postulations of Feller and Sternberg.

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5. Bielschowsky, Max, and Unger, Ernst: Syringomyelie mit Teratom-und extramedullärer Blastombildung, J. f. Psychol. u. Neurol. **25**:173, 1920.

6. Henneberg, R.: Ueber Geschwülste der hinteren Schliessungslinie des Rückenmarks, Berl. klin. Wchnschr. **58**:1289, 1921; Rückenmarksbefunde bei Spina bifida, Monatschr. f. Psychiat. u. Neurol. **47**:1, 1920.

7. Mackay, Roland P., and Favill, John: Syringomyelia and Intramedullary Tumor of the Spinal Cord, Arch. Neurol. & Psychiat. **33**:1255 (June) 1935.

REGENERATION OF POSTERIOR ROOT FIBERS IN THE CAT

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CHICAGO

Regeneration of sectioned posterior root fibers may be discussed from two points of view: regeneration in a proximal direction from the dorsal root ganglion and regeneration in a distal direction from possible cells of origin in the spinal cord.

The question of regeneration in a distal direction from cells of origin in the spinal cord is intimately related to the problem of the existence of efferent fibers in the posterior root. This question has been thoroughly reviewed by Sherrington [1] and more recently by Hinsie; [2] it would be superfluous to discuss the subject here. While it is generally agreed that there are efferent fibers in the posterior roots of certain lower forms, such as Amphioxus, Petromyzon, Pristiurus, Myxine and amphibians, birds and reptiles, the presence of such fibers in the posterior roots of mammals is still the subject of controversy, with an imposing array of workers on either side. It follows as a corollary that if there are no efferent fibers in the posterior root all fibers proximal to a section will undergo degeneration, and possibly regeneration. If there are efferent fibers in the posterior root all fibers proximal to the section will not show these phenomena, and, in addition, some fibers distal to the section should undergo these changes.

The question of degeneration and regeneration in a proximal direction is less controversial. Ranson,[3] in a cat, seventy-four days after section of a root, observed that up to the point where the root enters the cord the degenerated posterior root was crowded with fine axons which resembled normal fibers. Beyond this point only a few fine axons extended through the entering root into the cord. Ranson observed the same condition in two other cats fifty-one and seventy-five days

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Read before the Chicago Neurological Society, May 16, 1935.

From the Department of Nervous and Mental Diseases, the Northwestern University Medical School.

1. Sherrington, C. S.: On the Question Whether Any Fibers of Mammalian Dorsal Afferent Roots Are of Intraspinal Origin, J. Physiol. **21**:209, 1897.

2. Hinsie, J. C.: Are There Efferent Fibers in the Dorsal Roots? J. Comp. Neurol. **59**:118, 1934.

3. Ranson, S. W.: Lissauer's Tract in the Cat, J. Comp. Neurol. **23**:272, 1913.

after section of the root. Tower [4] sectioned the posterior roots in four cats. They were killed after four, six and twelve months, respectively. After four months she observed in the central stump numerous fine fibers, from 2 to 3 microns in diameter, the majority being unmyelinated; after a year most of these were myelinated. Some of the fibers penetrated the spinal cord along the blood vessels; none penetrated through the zone of root entry. Cate [5] stated that regenerating fibers are seen only as far as the entrance of the root into the cord. This writer also described small bundles of regenerating fibers in the septum which separates Lissauer's tract from the spinal cord; he never observed penetration of regenerating fibers into the spinal cord.

Experimental Data in Studies on Regeneration of Posterior Nerve Roots

Cat	Roots Sectioned	Duration, Days	Stains
1	Cervical 2, 3, 4, 5, 6, 7, 8 Thoracic 1, 2, 3, 4	374	Davenport; Weil; cresyl violet; Van Gieson; osmic acid; Perdrau; Holzer
2	Cervical 2, 3, 4, 5, 6, 7, 8 Thoracic 1, 2, 3, 4, 5, 6	26	Weil; Davenport
3	Cervical 2, 3, 4, 5, 6, 7, 8 Thoracic 1, 2, 3, 4	33	Weil; Davenport
4	Cervical 2, 3, 4, 5, 6, 7, 8 Thoracic 1, 2, 3, 4, 5	61	Weil; Davenport
5	Cervical 2, 3, 4, 5, 6, 7, 8 Thoracic 1, 2, 3, 4	86	Weil; Davenport
6	Cervical 2, 3, 4, 5, 6, 7, 8 Thoracic 1, 2, 3, 4	132	Weil; Davenport
7	Cervical 2, 3, 4, 5, 6, 7, 8 Thoracic 1, 2, 3, 4	111	Weil; Davenport

REPORT OF EXPERIMENTS

The material used for the present studies on regeneration consisted of seven cats in which some of the posterior roots were sectioned by Dr. Loyal Davis. The accompanying table indicates the roots cut and studied, the duration of time after section and the stains used.

Cat 1 lived three hundred and seventy-four days after section of eleven pairs of posterior roots. Sections of nerves from this cat were stained by the methods of Nissl, Van Gieson, Perdrau, Holzer, Davenport and Weil and with osmic acid. The sections will be described.

In preparations from this animal stained with cresyl violet (fig. 1), there were seen in the roots distal to the pia-glial limiting membrane densely packed, elongated nuclei arranged in strands. These were the nuclei of the protoplasmic bands of Büngner. Proximal to the pia-glial limiting membrane these bands did not appear; instead, there were many scattered glia nuclei.

4. Tower, S.: A Search for Trophic Influence of Posterior Spinal Roots on Skeletal Muscle, with a Note on the Nerve Fibers Found in the Proximal Stumps After Root Section, Brain **54**:99, 1931.

5. ten Cate, J.: Befunde nach der experimentellen Isolierung eines Rückenmarksabschnittes, Arch. néerl. de physiol. **17**:149, 1932.

In sections from the same animal stained by Van Gieson's method (fig. 2), there also appeared marked differences between the part of the root proximal to and the part distal to the pia-glial membrane. The distal part consisting largely of Büngner's protoplasmic bands, stained red, as is characteristic of connective tissue. Many darkly staining nuclei were seen in this region. Proximal to the point where the root enters the cord the picture was different. The tissue took a yellowish stain, and Büngner's bands were no longer present; instead, there were sparsely scattered oval nuclei.

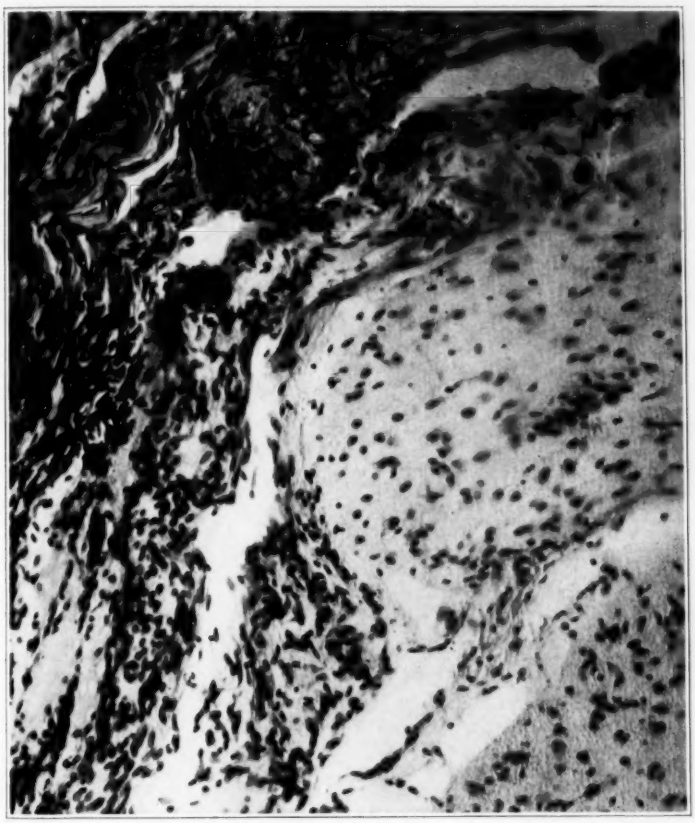

Fig. 1.—Photomicrograph (cresyl violet; × 375) of a degenerated eighth cervical posterior root, three hundred and seventy-four days after section. Distal to the pia-glial limiting membrane there are the densely packed nuclei of Büngner's bands; proximal to the membrane Büngner's bands are absent, and there are numerous glia nuclei.

Sections from cat 1 which were stained by Perdrau's method for connective tissue (fig. 3) showed that the part of the cord normally containing incoming fibers from the posterior root had been replaced by a dense meshwork of connective tissue. This connective tissue network was seen also in the extraspinal part of the root. The connective tissue fibers penetrated the limiting membrane in

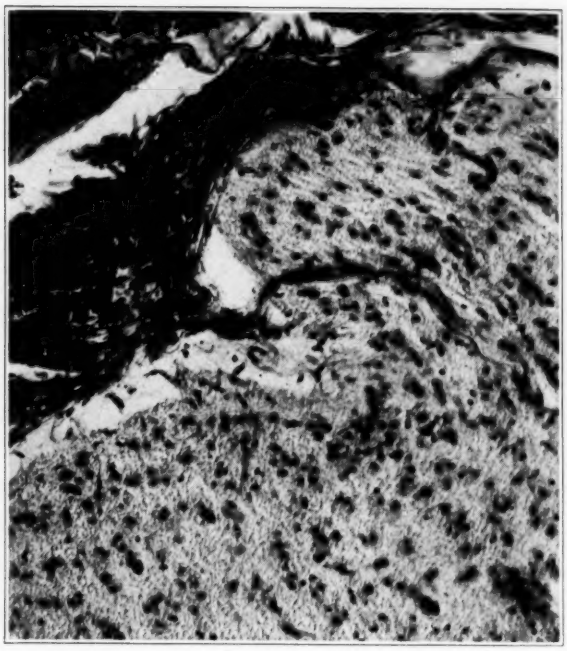

Fig. 2.—Photomicrograph (Van Gieson; × 375) of an eighth cervical posterior root, three hundred and seventy-four days after section. An abrupt change in appearance occurs at the pia-glial limiting membrane. Distal to the membrane there is a dense meshwork of connective tissue; proximally glia nuclei lie in a loose network of fibers.

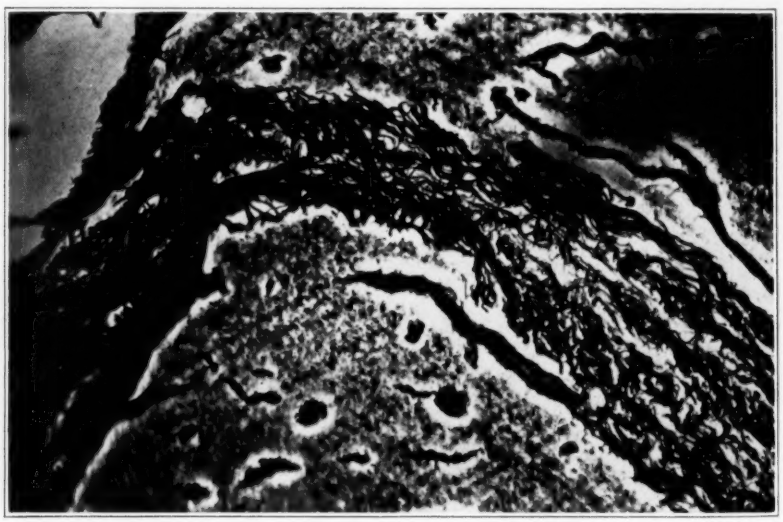

Fig. 3.—Photomicrograph (Perdrau's method for connective tissue; × 375) of a degenerated first thoracic posterior root, three hundred and seventy-four days after section. The part of the cord normally containing the incoming posterior root fibers is replaced largely by argentophilic connective tissue.

Fig. 4.—Photomicrograph (Holzer's stain; × 375) of a degenerated seventh cervical posterior root, proximal to the pia-glial limiting membrane. The posterior root fibers normally present are replaced by neuroglia fibers. An occasional astrocyte is seen.

Fig. 5.—Photomicrograph (osmic acid stain; × 375) of a regenerating first thoracic posterior root, three hundred and seventy-four days after section. Intact myelin sheaths are present.

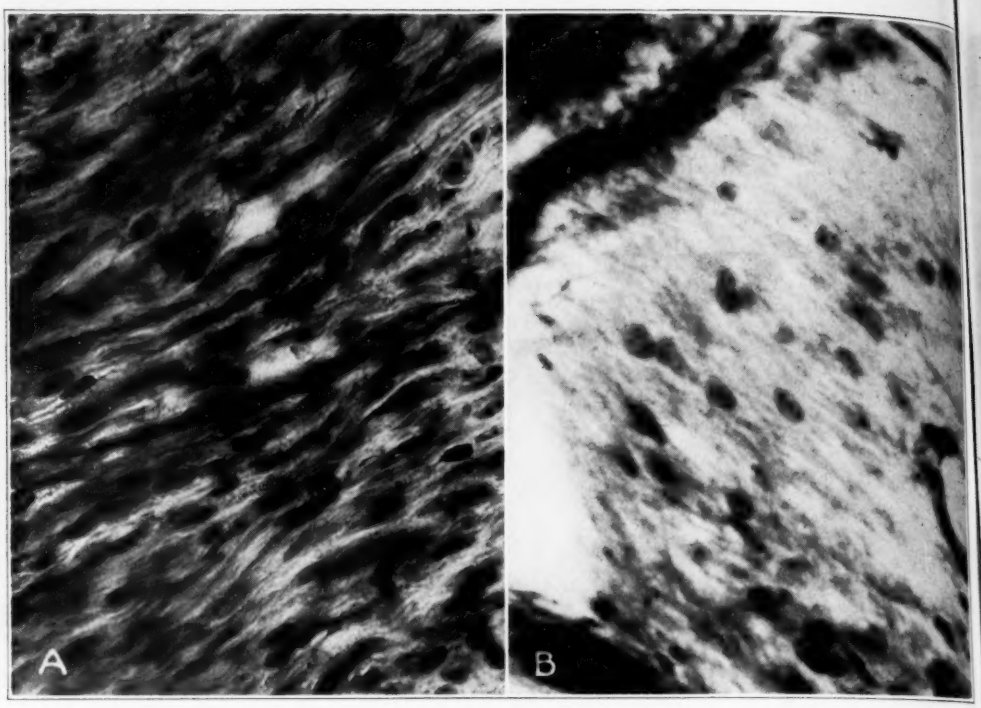

Fig. 6.—Photomicrographs (Davenport stain; × 650) of a degenerated eighth cervical posterior root, three hundred and seventy-four days after section, (*A*) just distal to the pia-glial limiting membrane showing nerve fibers lying in the densely formed protoplasmic bands of Büngner, and (*B*) proximal and adjacent to the pia-glial limiting membrane. No fibers pierce the pia-glial limiting membrane. Instead, astrocytic nuclei lie in a loose network of connective tissue.

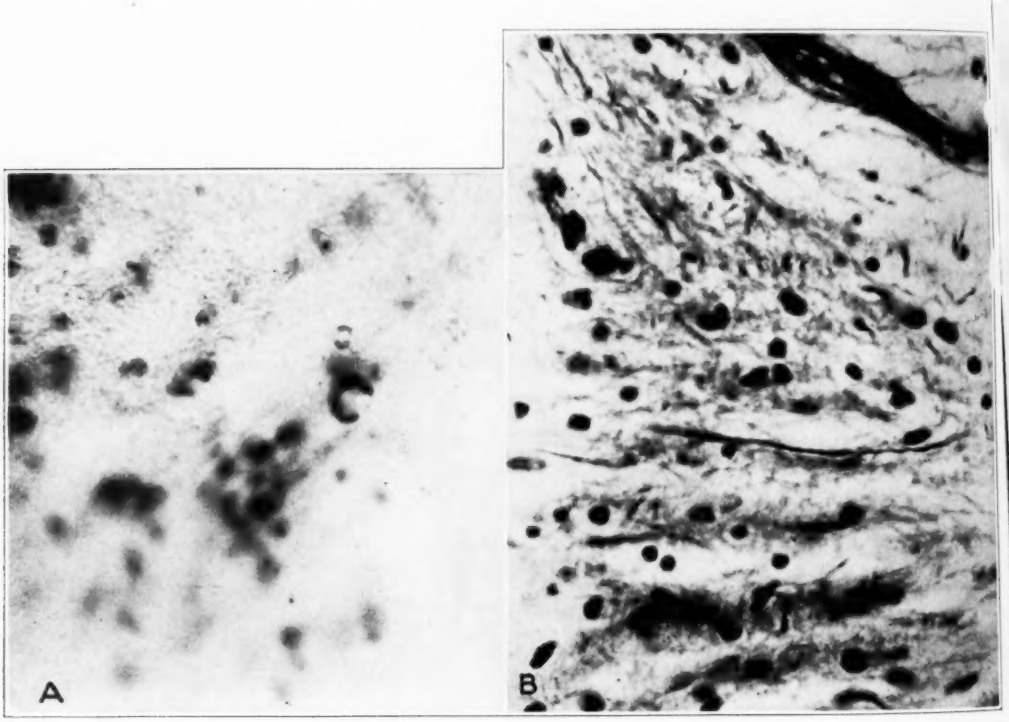

Fig. 7.—Photomicrographs (× 650) of a degenerated portion of the eighth cervical posterior root, three hundred and seventy-four days after section, at some distance proximal to the pia-glial limiting membrane. *A* (Weil stain) shows the presence of intact myelin sheaths, and *B* (Davenport stain) the presence of fine nerve fibers.

large numbers, and the meshwork continued into the cord, replacing incoming posterior root fibers. Sections stained by Holzer's method for glia fibers (fig. 4) showed that the portion of the root proximal to the pia-glial limiting membrane contained a dense network of thick glial fibers. Occasionally a large astrocyte was present in the meshes of these fibers.

Sections of the extraspinal portion of the roots from the same animal stained with osmic acid (fig. 5) showed numerous small or medium-sized medullary sheaths of regenerating fibers. These parts, stained by Davenport's method (fig. 6*A*), showed numerous regenerating fibers, none of which pierced the pia-glial limiting membrane (fig. 6*B*).

Although in this animal no fibers or medullary sheaths were observed piercing the pia-glial limiting membrane or in the regions just proximal and adjacent to

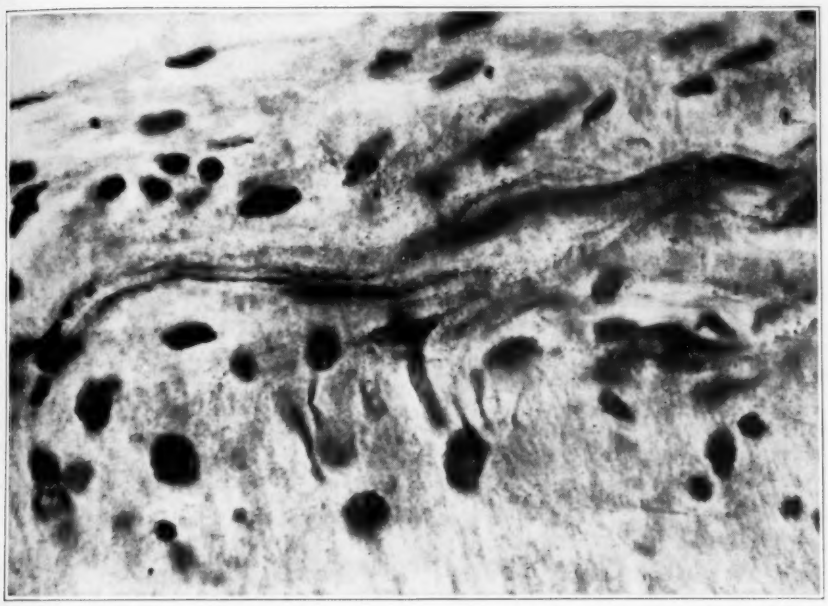

Fig. 8.—Photomicrograph (Davenport stain; × 650) showing fine nerve fibers entering the cord near a blood vessel.

the membrane, intact fibers and myelin sheaths were seen in the posterior root at some distance proximal to the membrane. These are illustrated in figure 7. The nature of these fibers remains obscure. They may be dendrites of the cells of Clarke's column or dentrites from the cells forming the comma tract of Schultze. It is hardly likely that they are efferent fibers; if they were, one should be able to trace them to and through the pia-glial limiting membrane. In these sections, however, they could not be traced. It is possible that these fibers may have entered the cord with a blood vessel, as occasionally intact fibers were observed along the blood vessels entering the cord (fig. 8).

In the remaining animals (twenty-six, thirty-three, sixty-one, eighty-six, one hundred and eleven and one hundred and thirty-two days after section of the root), only the intraspinal parts of the roots were available for study. These parts were

studied bilaterally at each level at which the roots were sectioned. The results confirmed those observed in cat 1 (three hundred and seventy-four days after section). No posterior root fibers were seen piercing the pia-glial limiting membrane and entering the cord.

SUMMARY

This study of regenerating posterior root fibers in the cat discloses some important differences between the part of the root distal to and the part proximal to the pia-glial limiting membrane. Distal to the pia-glial limiting membrane there are protoplasmic bands of Büngner and regenerated nerve fibers. Proximal to the pia-glial limiting membrane instead of protoplasmic bands of Büngner one sees glia nuclei in a loose network of glia fibers. No regenerating fibers are seen piercing the pia-glial limiting membrane into that part of the root lying proximal to it. Occasionally a regenerated fiber may find its way into the cord in association with a blood vessel. In the part of the degenerated root lying proximal to the limiting membrane and at some distance proximal to it there are intact fibers and medullary sheaths.

CONCLUSIONS

Posterior root fibers in the cat regenerate up to the pia-glial limiting membrane; none enter the cord in the zone of root entry.

A few regenerated fibers may enter the cord with blood vessels.

The significance of intact fibers and sheaths observed in the portion of the degenerated root proximal to and at some distance from the pia-glial limiting membrane is difficult to estimate. They may be regenerated fibers that entered the cord with blood vessels; they may be dendrites of the cells of Clarke's column; it is even possible that they may be efferent fibers which have cells of origin in the cord and do not degenerate after section of the posterior root. In the latter case, however, one would expect to see them piercing the pia-glial limiting membrane; this was not observed in these sections.

Dr. Arthur Weil gave valuable assistance and advice in this work.

MENINGO-ENCEPHALOMYELITIS NEONATORUM

ANATOMIC REPORT OF A CASE

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A satisfactory classification of the large group of congenital and early infantile diseases of the brain, Brissaud's *encéphalopathies infantiles,* does not as yet exist, despite a vast accumulation of reports of cases and descriptions published during the past seventy years.

A number of more or less well defined syndromes, such as amaurotic idiocy and Schilder's disease, have been delineated, it is true, but there remains a motley assortment of disturbances—traumatic, inflammatory and degenerative. These are generally designated as infantile or congenital encephalitis in such a loose way that "encephalitis" has scarcely any clinical or anatomic significance in the realm of diseases of the new-born. All this bespeaks a lack of understanding of the etiology and pathogenesis, on which some light may be shed by the observations in the case to be reported.

REPORT OF CASE

History.—A girl, aged 7 weeks, was admitted to the Presbyterian Hospital, to the service of Dr. Clifford G. Grulee, in a convulsive state and died eight hours after admission. The only history on record was that of a "cold" of one week's duration, with fever for five days and convulsions for twenty-four hours.

Examination.—The infant weighed 8 pounds and 11 ounces (3,941 Gm.). The temperature was 101.6 F. Convulsive twitchings and clonic spasms of the arms and legs, associated with opisthotonos, were present. The spinal fluid was xanthochromic; there were 880 red cells per cubic millimeter, some of which were degenerated, and 30 white cells; the total protein content was 1,260 mg. per hundred cubic centimeters, and the Wassermann reaction of the fluid was reported to be "unsatisfactory." The hemoglobin reading of the blood was 60 per cent; the erythrocyte count, 3,580,000, and the number of leukocytes, 54,000, 23 per cent of which were neutrophils, 50 per cent small mononuclears, 10 per cent large mononuclears, 3 per cent eosinophils, 2 per cent basophils, 2 per cent normoblasts and 3 per cent unclassified cells. The bleeding time was slightly prolonged, to seven and one-half minutes, and the clotting time was seven minutes. There was a marked decrease in the number and an increase in the resistance of the platelets. The serum calcium content was 9 mg. per hundred cubic centimeters, the plasma phosphorus content 8.7 and the carbon dioxide-combining power 38.6 cc.

Roentgenologic examination revealed increased markings involving the upper half of the right lung. Increased density back of the epiphysial lines of the long bones was also noted, with decreased density of the bone in these regions. These

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changes were not those of syphilis but suggested rather a nutritional disturbance. The Wassermann and Kahn tests of the blood were not made.

Clinical Diagnosis.—The clinical diagnosis was cerebral damage arising from a birth injury, bronchopneumonia and, possibly, von Jaksch's anemia.

Necropsy (Dr. E. M. Barton).—There were no significant changes other than those in the nervous system except typical bronchopneumonia, which appeared to be the cause of death. Nothing was observed grossly or microscopically to substantiate the tentative clinical diagnosis of a blood dyscrasia, and it is especially noteworthy that not the slightest indication of congenital syphilis was observed in any organ of the trunk or within the long bones.

Gross Appearance of the Brain: After hardening in a solution of formaldehyde the brain weighed 420 Gm. The leptomeninx of the convexity of the brain was somewhat thickened and opaque, with the appearance of leptomeningitis fibrosa. This was especially marked over regions where changes were apparent on the underlying surface of the brain. The meninges and the vessels of the base were normal. Most of the midbrain and isthmus and the upper portion of the pons had been cut away, but what remained of this portion of the brain stem appeared normal, except for a region of speckled yellow discolorations over the lateral aspect of the upper portion of the midbrain on the right side, including the peduncle. The cerebellum was normal in external appearance, as was the brain stem from the middle of the pons to the lower part of the medulla. No changes were seen on section of the cerebellum through the dentate nuclei. Cut surfaces of the pons and the medulla were likewise normal. The aqueduct of Sylvius was small and irregular but patent. The optic nerves and chiasm and the roots of the cranial nerves from the fifth to the twelfth were normal.

There was no abnormality of development of the cerebral hemispheres. The principal gyri and sulci were present and of normal size and design except where they were disturbed by the presence of lesions near the surface. These changes were patchy and consisted mostly of a yellowish softening of the gyri. In some regions this had led to localized atrophy of the convolutions, with disfiguration of the brain. The antero-inferior portion of the right frontal lobe was thus affected and was definitely smaller than the opposite frontal pole. The posterior end of the left middle frontal convolution was similarly affected, uncovering the anterior portion of the insula. The temporal poles on both sides were easily indented by palpation, as though they were the walls of a sac; the right was distinctly smaller than the left. A region of marked change of this kind was seen in the occipital lobe and the inferior parietal lobule on the left. This had produced a furrow, 1.5 cm. deep, extending laterally from the parieto-occipital fissure to the gyrus supramarginalis and gyrus angularis (fig. 1). Smaller round yellowish white discolorations of the cortex were present in various regions, ranging from 1 to 3 mm. in diameter.

On frontal section at the level of the tuber cinereum, definite enlargement of the entire ventricular system was observed. The left lateral ventricle measured 2.5 by 1 cm. in its greatest dimension and the right 1.5 by 1 mm. The third ventricle was 16 mm. in the vertical and 5 mm. in the transverse diameter. The foramina of Monro were patent. The region of the right internal capsule, the lateral portion of the thalamus, the entire putamen and caudate nucleus and much of the globus pallidus were completely destroyed. Part of this region had undergone a change resembling caseation and was occupied by a white cheesy material. Elsewhere, the affected region was soft, yellow and somewhat spongy in places. In the most external part of the area, just beneath the cortex of the insula, was a cavity 2.5 cm. long and 3 mm. wide, partly filled by delicate trabeculae of soft

spongy tissue. A second cavity in the left hypothalamic region extended laterally beneath the lenticular nucleus and broke into the third ventricle mesially. This was occupied by a soft yellow material. The regions in question were not well circumscribed or discrete but appeared to invade the surrounding tissue diffusely.

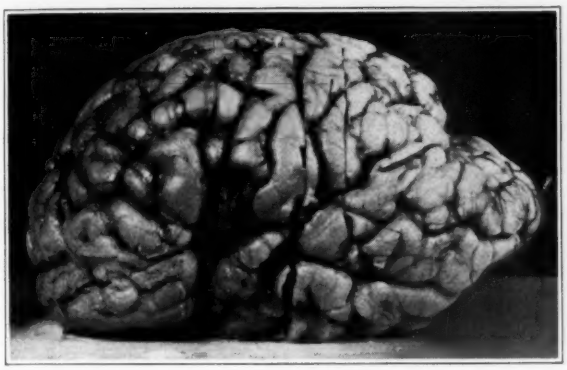

Fig. 1.—Photograph of the left cerebral hemisphere, showing convolutional deformities.

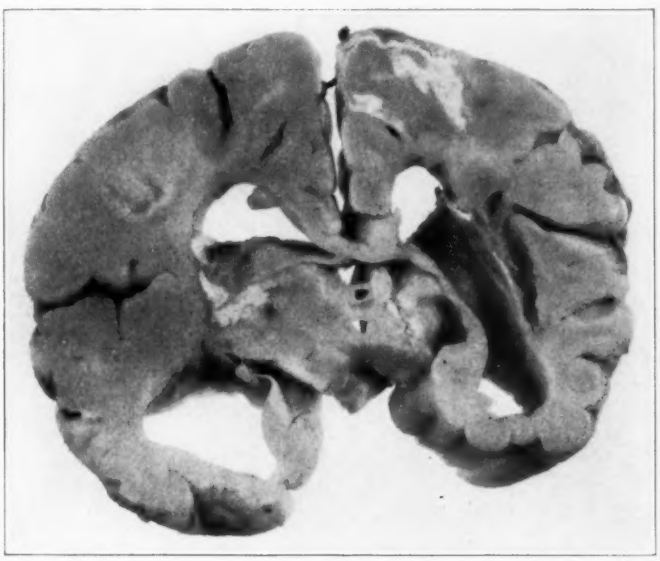

Fig. 2.—Photograph of a frontal section at the level of the massa intermedia, showing enlargement of the ventricles, colloid and caseous areas of necrosis, regions of softening and extensive calcification.

The necrosis extended up to the ventricular surface, and in many places the ventricular wall was speckled with small yellow patches or presented irregular regions of the same white caseous appearance seen in the softened areas. In places these projected slightly into the ventricle, roughening the surface. The tip of the

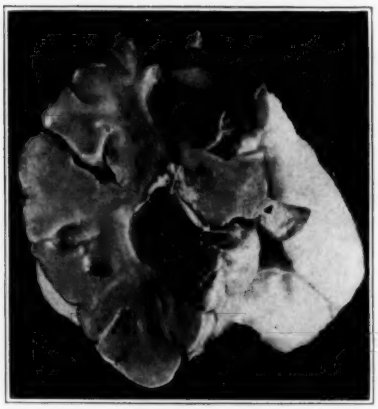

Fig. 3.—Photograph of a frontal section through the occipital lobe and the midbrain, showing periventricular lesions and destruction of the peduncle.

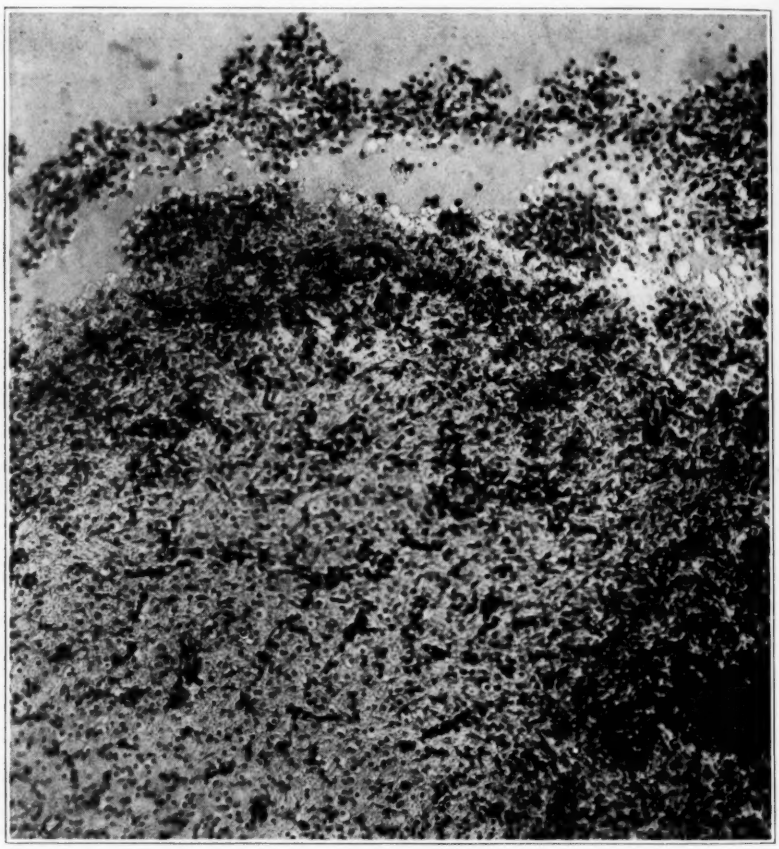

Fig. 4.—Photomicrograph of a necrotic area, showing colloid change in the upper portion of the field, with surrounding cellular and vascular reaction, and calcification to the right. Hematoxylin and eosin stain.

descending horn of the right lateral ventricle was partly filled by soft spongy tissue, like that described in the cavity. In addition, tiny yellowish flecks were seen here and there over the cut surface, which otherwise appeared normal. In the left superior temporal convolution was a linear white caseous area just beneath the pia, less than 1 mm. wide.

In a frontal section anterior to the peduncles and immediately anterior to the superior colliculi (fig. 2), there was similar dilatation of the ventricular system, as well as several large regions of necrosis and softening resembling those already

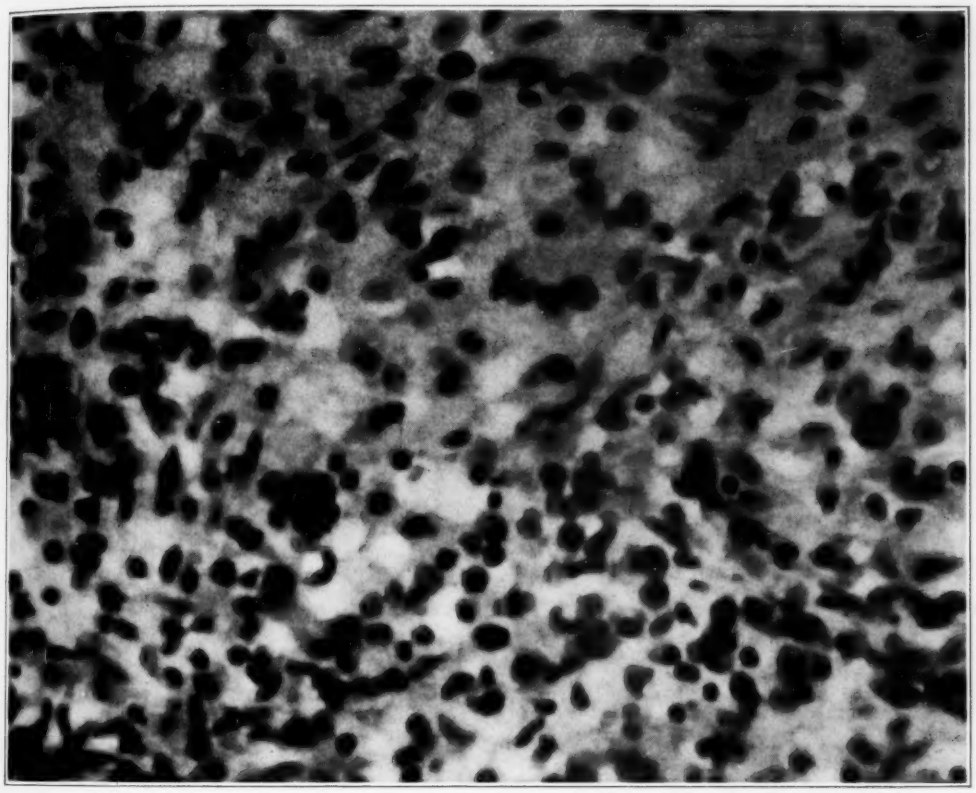

Fig. 5.—Photomicrograph showing glial and fibroblastic hyperplasia, with infiltration with plasma cells. Remnants of ganglion cells are incrusted with calcium. Hematoxylin and eosin stain.

described. One of these areas involved the pulvinar of the thalamus on the left. On the right the entire pulvinar, the lateral and medial geniculate bodies and the lateral aspect of the tegmentum of the midbrain and peduncle were completely destroyed and replaced by a shrunken yellow substance. In the upper part of the postcentral gyrus was a region, 2 by 1.5 cm., partly white and caseous and partly firm and with the glistening blue-white appearance of milky, opalescent glass. This alteration extended up to the meninges, which were observed to be thickened where the lesion came in contact with them. There was a cavity filled with

spongy tissue just lateral to the ventricle and running parallel with it. The intervening tissue had a soft, succulent appearance. Posterior to the peduncle the body and descending horn of the lateral ventricle communicated by a passage 6 mm. in width. On cut surfaces extending far forward in the frontal lobe and backward in the occipital lobe were disseminated regions of softening, some minute, others as large as 2 cm. in diameter. Most of these exhibited a change of the same white caseous type seen elsewhere and involved the basal nuclei, cortex and white

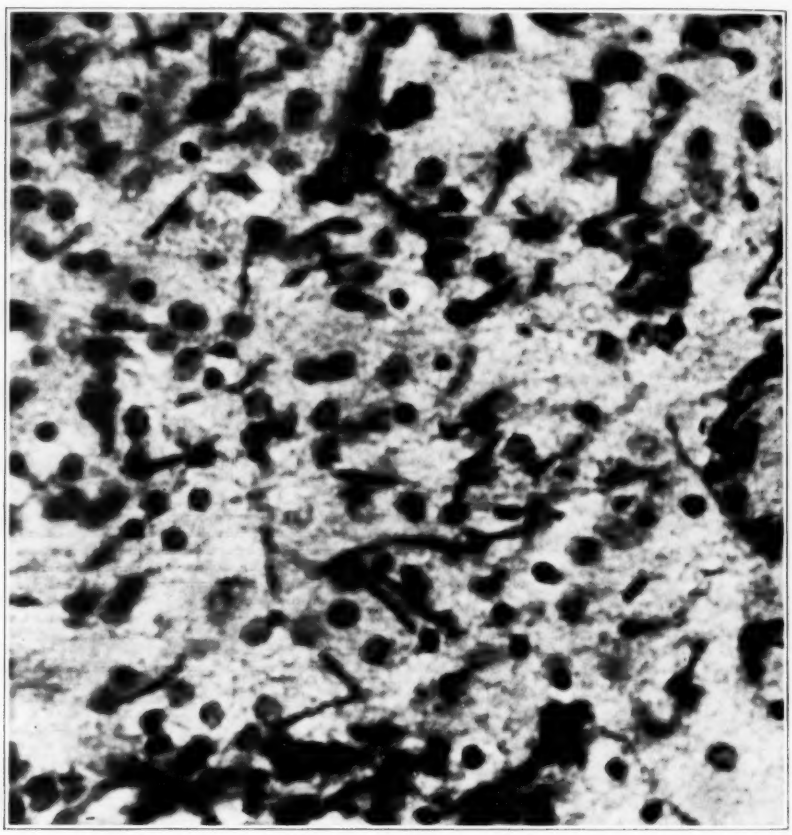

Fig. 6.—Photomicrograph showing rod cells. Iron hematoxylin stain.

matter indiscriminately. In many places they extended up to and into the ventricular surfaces or the pia, especially those in the occipital poles. In places the edges of the walls of the dilated ventricles were rigid, and there the ventricles were outlined by a band of firm, translucent gray-white tissue (fig. 3).

Microscopic Appearance of the Brain: In conformity with the gross appearance of the brain, the lesions were composed of irregular regions of necrosis, within which no vestiges of nerve cells or fibers remained, either in the Nissl or in the Bielschowsky preparations. The cores of these foci were made up of a structureless colloid substance, which stained pink with hematoxylin and eosin, yellow with

Nissl's stain and deep yellow-red with Van Gieson's. Within the substance were scattered groups of inflammatory cells and in many places a dustlike sprinkling of fine amorphous material, which stained deep blue with hematoxylin and was interpreted as calcium. At the borders of such regions there was a wide zone of inflammatory reaction, so extremely cellular and diffuse that at first glance it resembled tumor tissue (fig. 4). The enormous masses of cells of this inflammatory reaction were a mixed collection of fat granule cells, hyperplastic glia cells, proliferating fibroblasts and plasma cells (fig. 5). With scarlet red the fat

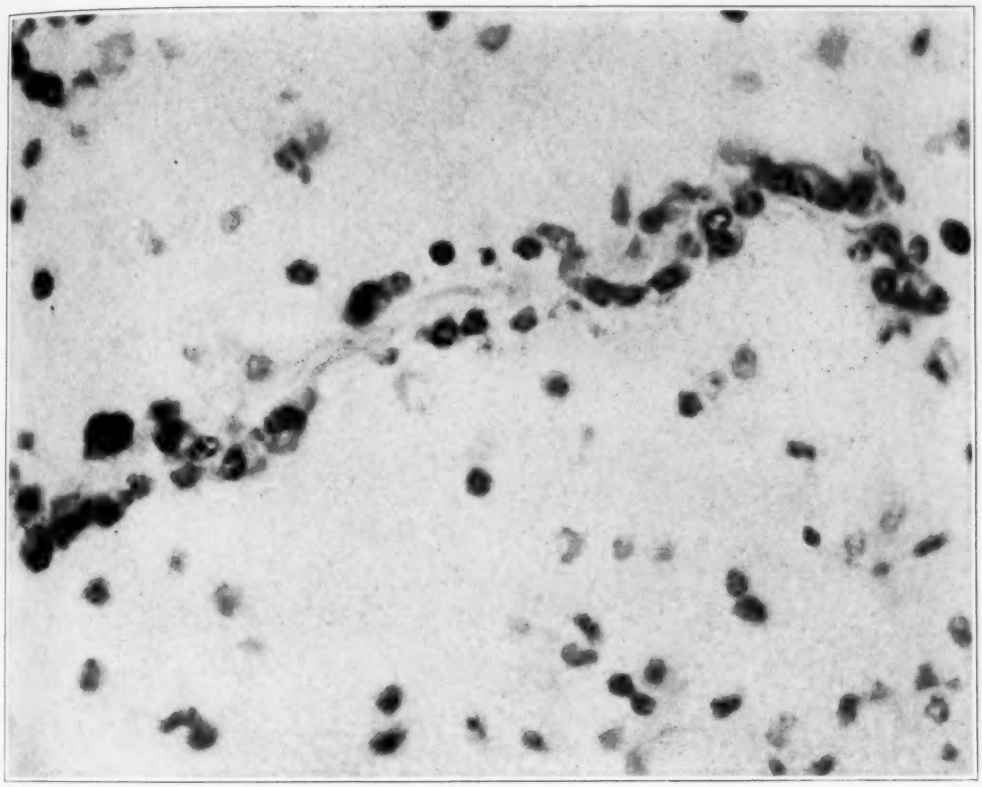

Fig. 7.—Photomicrograph showing plasma cells surrounding a newly formed capillary. Eosin and methylene blue stain.

granule cells were seen to be exceedingly numerous in and about the lesions; they crowded the adventitial spaces of the blood vessels in cuffs from ten to twelve cells deep. A diffuse increase of astrocytic and oligodendroglial nuclei was apparent in all sections but became greatly exaggerated in the vicinity of the lesions. Staining with the Alzheimer-Mann and Hortega methods demonstrated that in this locality the glia was also hyperplastic and presented the features of the *gemästete* type. Growth and sprouting of new capillaries were a prominent feature of the inflammatory zone, and there was notable proliferation of these

structures into the surrounding area. Immediately adjacent to the necrotic foci were a considerable number of rod cells (fig. 6). These were in close relation to the proliferating blood vessels and were apparently of mesodermal origin. The most striking feature of the exudative process was the huge number of plasma cells. Many were in the adventitial spaces of the capillaries, where they often formed complete sleeves (fig. 7); many were also seen scattered free in what remained of the parenchymatous tissue, often in massive accumu'ations (fig. 8). A number of the plasma cells were multinucleated.

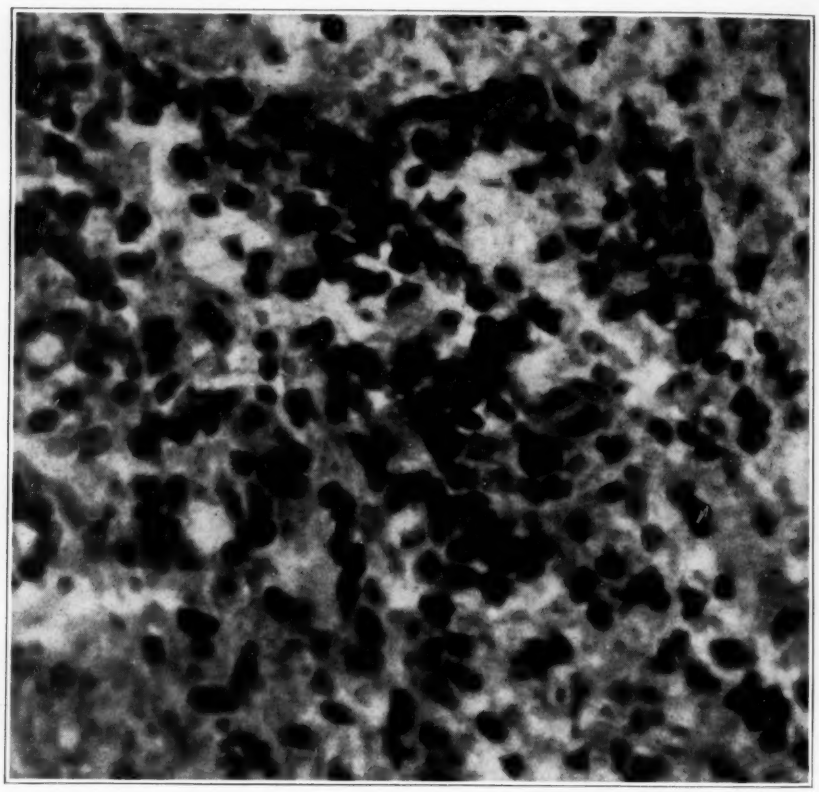

Fig. 8.—Photomicrograph showing massive parenchymatous infiltration of plasma cells. Thionin stain.

Few lymphocytes entered into the exudate. A large number of glial nodules were present in the inflammatory area. These were especially numerous near the periphery. In Van Gieson preparations there was distinct edema of the glial reticulum, and necrotic change of the colloid type appeared to begin as a deposit of small round globules of homogeneous material within the swollen glial reticulum. A large amount of amorphous material taking a dark blue stain with hematoxylin was observed here and there in the inflammatory region, sometimes in small and sometimes in massive accumulations. It was soluble in hydrochloric acid and had every appearance of a deposit of calcium. Incrustations of the same

substance coated what appeared to be the remains of a number of ganglion cells, as well as some of the rod cells (figs. 5 and 6). There was a marked tendency, variable from place to place, toward organization of the lesions, both from glial and from mesodermal sources. It was not possible to stain the neuroglial fibers by means of any of the usual stains, such as those of Weigert, Mallory or Holzer or the victoria blue, but in Hortega silver preparations a dense network of fine fibrils was clearly demonstrated. In Van Gieson preparations a variable but rather small amount of collagenous tissue was seen invading the destroyed areas

Fig. 9.—Photomicrograph showing a reticulum scar within a lesion. Davidoff's method.

from the neighborhood of the blood vessels. In all areas studied, however, there was a dense reticulum scar (fig. 9).

All lesions of the deeper portions of the parenchyma, whether of the basal nuclei, the central white matter or the deeper layers of the cortex, were essentially similar except for differences in the degree of necrosis, exudation or organization, depending on variations in the ages of the respective foci. In lesions which lay at the meningeal and at the ventricular surfaces there were also special noteworthy features. The leptomeninges of the cerebral hemisphere were everywhere markedly thickened and fibrotic, containing a great increase of both collagenous and reticular

fibrous tissue. They were also heavily infiltrated with plasma cells and large mononuclear cells and relatively slightly with lymphocytes. Beneath this reacting leptomeninx there were in many places narrow, superficial lesions of necrotizing inflammation, similar in all respects to those already described, which destroyed only the outer subpial layers of the cortex (fig. 10). Organization of such areas was accomplished by proliferation of fibroblasts not only from the adventitia of the blood vessels but from the meninges. The pia was closely adherent to the brain, and from it tufts of fibroblasts could be seen growing into the underlying brain. In this way, the line of cleavage between the brain and the pia was com-

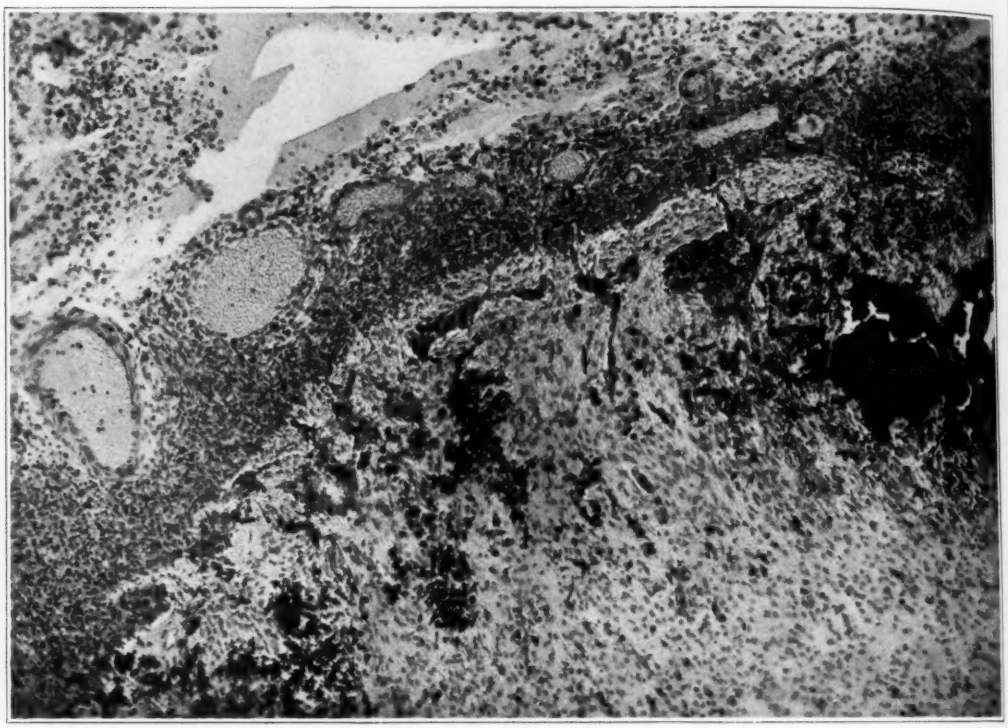

Fig. 10.—Photomicrograph (hematoxylin and eosin stain) showing hyperplastic infiltration of the meninges, with a superficial cortical lesion. The parenchyma is invaded by connective tissue from the meninges.

pletely obliterated, except that the frequent deposits of calcium in these lesions tended to stop short at this boundary. Isolated, scattered crystals of calcium salts or soaps were, however, present in considerable numbers in the meninges. Nowhere was there any ependymal lining of the lateral ventricles; only a part of the wall of the third ventricle was covered by ependymal cells, which were in poor condition. Beneath these surfaces was a dense cellular reaction, from one-fourth to one-half the diameter of a low power field in width. For the most part this reaction was produced by gliosis, but many plasma cells were mingled with the glial forms (fig. 11). These tended to be arranged in rows just beneath the ependyma, where it still persisted, and also to occupy small glial papillae, which

projected into the ventricles in many places. Extensive regions of necrosis occurred in this periventricular area of gliosis. These were usually the site of dense calcium deposits, the plaques lying bare to the ventricles. In addition, perivascular infiltrations of the kind described elsewhere, as well as glial nodules, were present in the periventricular fields.

Microscopic Changes in the Spinal Cord: In sections from many cervical, dorsal and lumbosacral levels the usual change was moderate meningeal thickening associated with some infiltration by plasma cells and macrophages, but much less

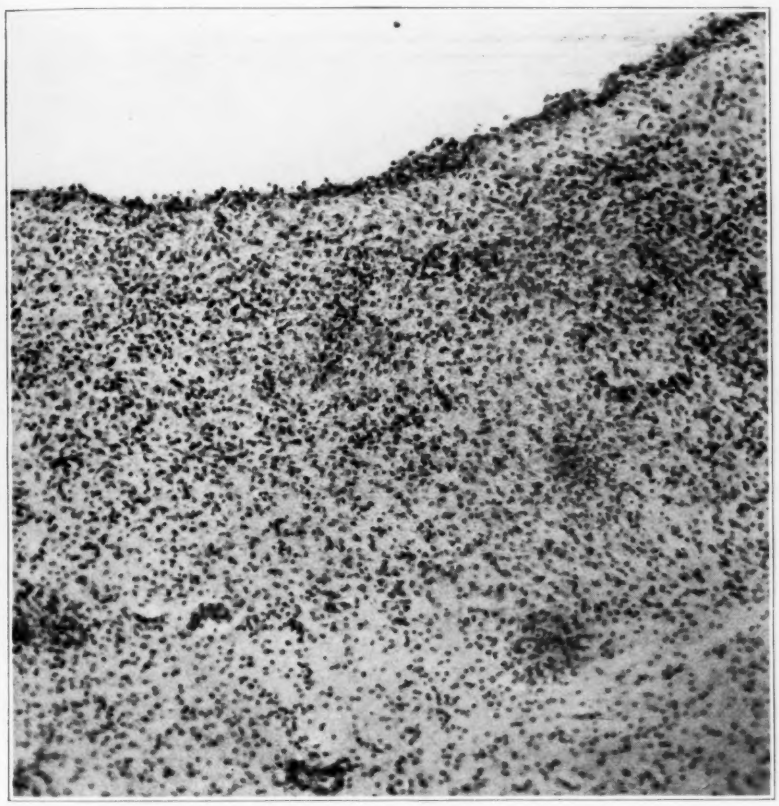

Fig. 11.—Photomicrograph of the ventricular surface, showing destruction of ependyma, periventricular gliosis and infiltration and glial nodules. Thionine stain.

marked than that of the cerebral pia-arachnoid. In the cord itself was a diffuse increase of glia nuclei, with occasional heaping up of these nuclei into nodules. In one region in the upper thoracic portion of the cord there were marked alterations (fig. 12). The lower portion of the cord on the right side was entirely destroyed, and the damaged area was replaced by myriads of glial and fibroblastic nuclei, rod cells and some plasma cells, the latter lying chiefly in the capillary walls. These changes extended without sharp demarcation dorsally and into the opposite side of the cord. The anterior horn cells on the right side were com-

pletely obliterated; on the left they were disappearing. The whole of the affected region was traversed by numerous greatly thickened vessels. In cross-section many of these vessels appeared as whorls of fibroblasts without a lumen, and they were frequently partly hyalinized. In this focus were no regions of softening or colloid change and only a few isolated amorphous deposits of calcium. The meningeal thickening and infiltration were much greater at this point than elsewhere, and in this region the meninx was fused to the cord on the affected side, with invasion of the parenchyma by fibroblasts, which carried a scaffolding of reticulum and collagen fibers.

Other Lesions: In addition to the essential lesions described, a number of special and general features were worthy of mention. In the pons and medulla was a diffuse increase of glia nuclei, with a few nodules. The meninges of these

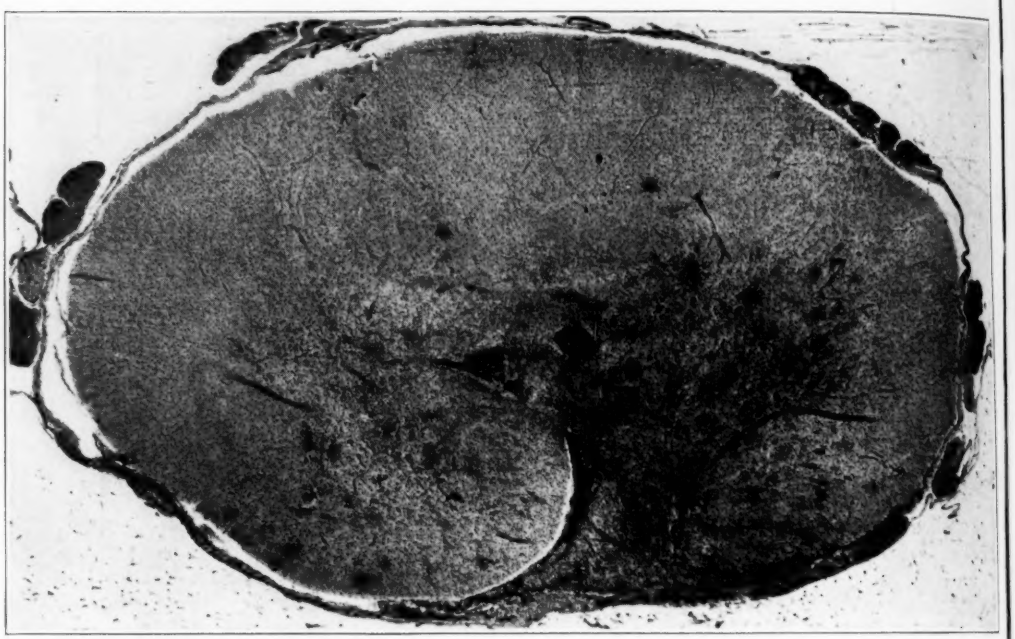

Fig. 12.—Photomicrograph showing a focus of destruction in the spinal cord. Hematoxylin and eosin stain.

structures were thickened and infiltrated, but more moderately than the pia-arachnoid of the hemispheres. In the pons also there was proliferation of the adventitial sheaths of the vessels, with numerous plasma cells in the perivascular spaces of many. The cerebellum was essentially normal histologically, and no binucleate Purkinje cells were noted. There were marked changes in the ganglion cells of the entire cortex. Generalized alterations of the ganglion cells of the basal ganglia and brain stem were likewise evident, decreasing in intensity from the higher to the lower levels, so that in the spinal cord they were slight. In the cortex, where they were greatest, these changes were typical of Nissl's "water change." Sections of the pons, medulla and spinal cord stained by Weil's method for myelin sheaths showed no patches of demyelinization or any abnormality of the principal myelin architecture of these parts.

except for marked pallor in the staining of the left pyramidal tract. In sections from the anterior and posterior parts of the brain stained with scarlet red, large amounts of fat were observed in the glia cells of the white matter in the neighborhood of the ventricles. In most of the cells the fat was evenly dispersed in the form of fine droplets, and the cells themselves were distributed in the fields with regularity, not grouped into clumps. These interstitial fat cells were regarded not as pathologic but as normal *Aufbau* phenomena concerned with myelinogenesis.

There were generalized changes in the arteries everywhere in the brain and spinal cord, consisting of thickening of all coats, especially the intima and adventitia. No special tendency to thickening or fragmentation of the internal elastic membrane was shown with the Weigert elastic fiber stain, and there was no thrombosis. Obvious alterations were present also in almost all capillaries, aside from the new formation already mentioned. These changes were chiefly retrogressive. The walls of the vessels stained deeply with eosin, and in general the capillaries appeared as shrunken cords rather than as tubes. The epithelium of the choroid plexus was of normal appearance, but in many places the connective tissue stroma was infiltrated with plasma cells.

The methylene blue and Gram stains revealed no organisms in the tissues. Numerous attempts to stain spirochetes by the methods of Levaditi, Jahnel, Dieterle and Kanzler yielded uniformly negative results.

COMMENT

Among the French observers who have dealt with the question of infantile encephalopathies there is a tendency, according to the scheme of Babonneix and Voisin,[1] to classify the cases under the general headings of: (1) developmental defects; (2) hydrocephalus; (3) porencephaly and pseudoporencephaly; (4) chronic meningitis, and (5) chronic meningo-encephalitis. Within the last division are included a number of anatomic specimens presenting various grades of parenchymatous degeneration and necrosis, with reactive changes in the glia, connective tissue and blood vessels. These are not clearly differentiated, on the one hand, from encephalopathies due to early circulatory (asphyxial) disturbances, so well described by Wohlwill,[2] to which may belong the cases reported by Alpers[3] under the new appellation of "diffuse progressive degeneration of the gray matter of the cerebrum," or, on the other hand, from congenital syphilitic meningo-encephalitis. Babonneix[4] was aware of the uncertainties involved and of the reasons for them, which lie chiefly in the circumstance that the same anatomic lesion may be common to distinct clinical or etiologic conditions and,

1. Babonneix, M., and Voisin, R., in Hutinel, J.: Les maladies des enfants, Paris, Asselin & Houzeau, 1909, vol. 5, p. 116.

2. Wohlwill, F.: Zur Frage der sogenannten Encephalitis congenita (Virchow), Ztschr. f. d. ges. Neurol. u. Psychiat. **68**:384, 1921; **73**:360, 1921.

3. Alpers, B. J.: Diffuse Progressive Degeneration of the Gray Matter of the Cerebrum, Arch. Neurol. & Psychiat. **25**:69 (March) 1931.

4. Babonneix, M.: Contribution à l'étude anatomique des encephalopathies infantiles, Encéphale **5**:310, 1910.

conversely, that similar states may produce diverse and variable anatomic pictures.

It cannot be doubted that the condition in the case reported in this paper represents a true inflammatory disease. There is evident a great similarity of the exudative changes and the mesodermal reactions to those of congenital syphilis, as described by Ranke,[5] who considered the picture characteristic. Moreover, necrosis with or without softening, while relatively uncommon in cases of congenital cerebral syphilis, is by no means unknown, as shown in the reports of Mathewson,[6] Beitzke,[7] Seikel [8] and Schmeisser.[9] Nevertheless, it is believed that one is justified in concluding that this case was not one of syphilis, although important clinical data on this point were, unfortunately, lacking. It is recognized that the failure to demonstrate spirochetes in the material does not conclusively exclude the possibility of syphilis, but it should be pointed out that the organisms are ordinarily observed with regularity in cases of congenital syphilis in which anything like the amount of damage present in this brain has been produced.

A more valid consideration is the complete absence of syphilitic alterations in other tissues, notably, the lack of periostitis, osteochondritis or hepatitis. It should also be mentioned that the changes in the spinal cord do not conform to those described for congenital syphilis by Toyofuku.[10]

Instances of nonsuppurative encephalitis, either congenital or in the new-born, in which, as in this case, the requirements of the modern morphologic criteria of inflammation are met are surprisingly seldom encountered in the literature. Among the best examples are the first of two cases recently reported by Diamond,[11] in which, except for the exudates, the changes were like those in the brain in the present case, and the one instance of congenital encephalitis included by Wohlwill [2] in his notable study of congenital encephalopathy, which was almost an exact duplicate of the case reported in this paper. The huge deposits

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5. Ranke, O.: Ueber Gewebsveränderungen im Gehirn luetischer Neugeborener, Neurol. Centralbl. **26**:112 and 157, 1907.

6. Mathewson, G.: Ueber einen Fall von congenitaler Syphilis ausgezeichnet durch ungewöhnliche Ausbreitung und Schwere der syphilitischen Erkrankung, Prag. med. Wchnschr. **20**:114, 1895.

7. Beitzke, H.: Ueber einen Fall von Erweichungsherden im Grosshirn eines kongenitalen syphilitischen Säuglings, Charité-Ann. **35**:382, 1911.

8. Seikel, R.: Ependymitis ulcerosa und Riesenzellenleber bei Lues congenita, Centralbl. f. allg. Path. u. path. Anat. **33**:337, 1922.

9. Schmeisser, H.: Ueber akute syphilitische Meningoencephalitis bei Neugeborenen, Beitr. z. path. Anat. u. z. allg. Path. **53**:151, 1912.

10. Toyofuku, T.: Die Veränderung am Rückenmark hereditär-luetischer Neugeborener, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **18**:31, 1910.

11. Diamond, I.: Encephalomalacia in Infants (Virchow's Interstitial Encephalitis), Arch. Neurol. & Psychiat. **31**:1153 (June) 1934.

of calcium, which were an outstanding feature in my case, were absent, but this is a quantitative, not a qualitative, difference. In general it can be said that the brains of young infants are prone to contain certain deposits of calcium salts of fatty acids in greater or less amounts whenever there have been severe retrogressive changes in the parenchyma from any cause. These are frequently described both in the form of amorphous precipitates and as incrustations of the cells. Wohlwill, too, noted the close resemblance of the changes in his case to those of congenital syphilis; he discarded the possibility of syphilis for much the same reasons as are given here. One feature in particular, which was common to the reaction in these two cases and to congenital syphilis, was the predominance of plasma cells in the exudates and the extensive dissemination of these elements through the tissues outside the adventitial spaces. The inference is drawn that in brains of infants of this age the pia-glial limiting membrane has not yet developed and offers no barrier to the wandering of these cells. It may be assumed in the same way that noxae have an easier access to the parenchyma and that this may account in part for the greater diffuseness of syphilis and encephalitis in infants, as compared with the same processes in more mature brains. Furthermore, the general and special similarities of the pictures in these two cases to each other and to congenital syphilis may be conceived as the expression of a more or less uniform mode of reaction of an immature brain to toxic-infectious agents of this order. In Wohlwill's case the disease process was undoubtedly congenital, since the infant died in three days. In my case the apparent age of some of the lesions makes it probable that the condition was also congenital, but since no proof for this assumption was at hand it was not so designated. If congenital the process must have started late in gestation, judging from the lack of developmental defects.

Almost every discussion of congenital or early infantile encephalitis during the past fifty years has been encumbered with Virchow's [12] concept of congenital interstitial encephalitis. What Virchow described and so designated were accumulations of gliogenous fat granule cells in the white matter of the brain and cord. As in the adult brain, this change is not specific, and there is no justification for regarding it as an anatomic entity with a specific pathogenesis, as Virchow did when he conferred on it the meaningful name "encephalitis." Many subsequent writers, such as Ceelen,[13] Schminke [14] and Diamond,[11] have

12. Virchow, R.: Congenitale Encephalitis und Myelitis, Virchows Arch. f. path. Anat. **38**:129, 1867; Ueber interstitielle Encephalitis, ibid. **44**:472, 1868; Encephalitis congenita, Berl. klin. Wchnschr. **9**:705, 1883.

13. Ceelen, W.: Ueber Gehirnbefunde bei Neugeborenen und Säuglingen (Encephalitis congenita Virchow), Virchows Arch. f. path. Anat. **227**:152, 1920.

14. Schminke, A.: Encephalitis interstitialis Virchow mit Gliose und Verkalkung, Ztschr. f. d. ges. Neurol. u. Psychiat. **60**:290, 1920.

reported under the traditional heading of Virchow's congenital or interstitial encephalitis cases in which there was little if any resemblance to what Virchow originally described, thus adding to the confusion. The term is ill conceived, represents nothing real and is misleading. One wishes with Wohlwill that it might be relegated to a position of mere historical interest.

CONCLUSIONS

A true infectious nonsuppurative meningo-encephalomyelitis exists in the new-born, an instance of which is described.

It is urged that such terms as encephalitis congenita, encephalitis neonatorum and the like be reserved for conditions of this type presenting the pathologic credentials of a true primary inflammation.

ADDENDUM.—Since the completion of this paper my attention has been directed by the recent report of Bucy and Buchanan [15] to the possibility that lead may be the etiologic agent in this case. This possibility, however, seems to be remote. While no anatomic descriptions of brains of infants of this age who died of lead poisoning are available for comparison, the widespread necrosis and extensive exudative reactions present in this case did not resemble the usual picture of acute lead encephalopathy as it is known in the adult brain, or even in the brains of infants somewhat older than this patient. Moreover, the roentgenologic appearance of the long bones was not that of saturnism. The narrow lines of density were noted not at the epiphyses but at some distance (about 0.5 cm.) back of them. It is scarcely conceivable that this could represent an antecedent deposit of lead in an infant of this age, nor would this be consistent with the acute, active nature of the process. Finally, quantitative analyses yielded 0.427 mg. of lead in 200 Gm. of brain tissue, as compared with 0.335 mg. in the same amount of brain tissue from an infant slightly younger, which was used as a control. This difference is not considered significant.

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15. Bucy, P., and Buchanan, D.: The Simulation of Intracranial Tumor by Lead Encephalopathy in Children, J. A. M. A. **105**:244 (July 27) 1935.

Abstracts from Current Literature

Physiology and Biochemistry

THE EFFECT OF AGE ON THE PLASMA CALCIUM CONTENT OF MEN. ESBEN KIRK, H. WILLIAM LEWIS JR. and WILLIAM R. THOMPSON, J. Biol. Chem. **111**: 641, 1935.

Up to the age of 85, the calcium content of the plasma of normal adult men undergoes no appreciable change. These data contradict the view that senile changes are due to increased concentration of calcium in the blood.

PAGE, New York.

STUDIES ON MAGNESIUM DEFICIENCY IN ANIMALS: VII. THE EFFECTS OF MAGNESIUM DEPRIVATION, WITH A SUPERIMPOSED CALCIUM DEFICIENCY, ON THE ANIMAL BODY, AS REVEALED BY SYMPTOMATOLOGY AND BLOOD CHANGES. HARRY G. DAY, H. D. KRUSE and E. V. MCCOLLUM, J. Biol. Chem. **112**:337, 1935.

Day, Kruse and McCollum studied the chemical and physiologic effects of extreme deprivation of magnesium and calcium in young dogs. They wished to ascertain whether under these conditions calcium and magnesium exhibited the antagonistic effects observed in lower animals. Of the phenomena following on restricted intake of calcium alone it is difficult for two reasons to give a clear description. First, most of the experiments were conducted with diets inadequate in several respects, particularly in vitamins, and, second, failure of appetite complicates the course of calcium deficiency. However, the principal signs and symptoms of calcium deficiency appear to be: foul, bloody diarrhea, progressive anorexia, loss of weight, epiphora, resorption of bone and hyperirritability of the nervous system, often eventuating in tonic-clonic convulsions.

Like calcium deficiency, deprivation of magnesium acts predominantly on the osseous and nervous systems, yet the effects in the two conditions bear no resemblance. Initial hypercalcification of the bones, due to calcium retention, which is the immediate response to magnesium deficiency, later gives way to normal deposition of calcium as the excretion of calcium is resumed. The nervous symptoms, hyperirritability, vasodilatation and tonic-clonic convulsions, which constitute tetany due to magnesium deficiency, represent a syndrome which is distinct from other forms of tetany. Hyperexcitability, particularly to auditory stimuli, increases until it is so pronounced that any undue noise may release convulsive seizures. Appetite is little altered throughout. If the period of survival is sufficiently long nutritive failure appears, but convulsions always kill the animals. The magnesium level of the serum undergoes prompt and rapid decline until it reaches an extremely low level, where it usually remains stationary. Conversely, the calcium and phosphorus contents, the p_H, the carbon dioxide capacity and chlorides remain normal.

With these two syndromes in mind, it is not difficult to ascertain points of similarity and difference which the states of magnesium and of calcium deficiency bear to each other. In animals deprived of both elements gastro-intestinal disturbances appeared in the form of severe obstipation, followed by severe diarrhea. Anorexia set in, with loss of weight. Marked osteoporosis was observed. Most of the animals were quiet and somnolent. The survival period was much longer than that in animals deprived of magnesium alone. Thus, the symptoms of deficiency of both magnesium and calcium partake predominantly of those of calcium deficiency, to the exclusion of the characteristics peculiar to magnesium deficiency.

Similarly, the blood changes differ from those characteristic of magnesium deficiency alone. In both types reduction in the magnesium content of the serum is shown, but the decrease in the amounts of calcium and phosphorus, the p_H and the carbon dioxide capacity and the increase in fibrinogen and chlorides displayed in magnesium and calcium deficiency are changes not seen in magnesium deficiency and probably are characteristic of calcium deficiency.

In order to judge the operation of an antagonism between calcium and magnesium, the study is limited to the consideration of symptoms and the data on blood. When the amount of either calcium or magnesium in the blood is sufficiently low the result is tetany. If deprivation of either element alone can bring this about, it would be expected that diminution of both would lead to more rapid onset and a more severe course. The symptomatology does not bear this out. The outstanding feature of deprivation of both calcium and magnesium is the absence of the appearance of the rapidly moving and fatal tetany characteristic of lack of magnesium. Tetany did not appear when changes in the contents of calcium and magnesium in the blood were indicative of it. The lack of calcium so counterbalanced the lack of magnesium that marked nervous symptoms rarely occurred. These data offered additional evidence in favor of the physiologic antagonism of calcium and magnesium.

PAGE, New York.

NOTE ON THE NATURE OF THE MOTOR DISCHARGE IN SHIVERING. D. DENNY-BROWN, J. B. GAYLOR and V. UPRUS, Brain **58**:233, 1935.

In this study the authors investigated the possibility of any existing relationship between the character, location and intensity of the movements in the muscular contractions of shivering in different regions of the body. For this purpose action currents were recorded by a Matthews oscillograph from the jaw, thorax, and biceps brachii and quadriceps femoris muscles concurrently. Shivering was induced by rapidly cooling the right upper and lower limbs by ice-water after the subject had been warmed until sweating had occurred. As the body temperature fell the first indication of the onset of shivering was usually an isolated twitch of the masseter muscle. The preliminary twitch was followed by others at variable intervals.

The intensity of shivering in any particular muscle group, together with the time of onset of shivering, appear to be related mainly to the amount of other contraction present. A muscle completely at rest shows early twitching and early fusion of twitches. A maintained posture, however slight, delays the onset of shivering. A voluntary movement in the more intense stages of shivering is followed by a greater outburst of shivering in the muscles concerned. Similarly, a deep breath is accompanied by partial or complete subsidence and is followed by increased shivering for an interval.

The twitching and the periods of rapidly repeated clonic contraction were not coincident in any two of the sites examined. The rate of rapid beating varies from time to time in any one muscle group, and when two muscle groups are beating concurrently there is no correspondence in their rates. The smaller muscles usually contract more rapidly, and movements of the hip and shoulder are slightly slower than those of the jaw or distal joints of the limbs.

The action currents of shivering muscle sooner or later become grouped into dense volleys, separated by quiet intervals corresponding to the regular alternation of the clonic beats. In each of these beats complete fusion of the action currents into one large electrical variation was not seen at any time, a feature which is in sharp contrast to the electromyogram of the reflex clonus of spastic muscle. The clonus of shivering and that resulting from damage to the pyramidal tract differ in the nature of the discharge and in their relationship to cold and to voluntary movement.

SALL, Philadelphia.

THE OCULAR PULSE. P. BAILLIART, Ann. d'ocul. **172**:701 (Aug.) 1935.

The ocular pulse caused by changes in the volume of the eyeball may be studied by using the manometer and the tonometer. Through this oscillometric procedure it may be possible to devise a method of measuring the intra-ocular arterial pressure. Using the kymograph of Gomez and Langevin, Bailliart studied the curve of tension of the eyeball at different times in the cardiac cycle. Useful information may also be gained in regard to the rapidity with which the blood flows toward the eye.

BERENS, New York.

SALIVARY CONDITIONED REFLEXES IN HEALTHY PERSONS AND PATIENTS WITH PSYCHOSIS AND THEIR COMPARISON WITH THE DATA OF CONSCIOUSNESS. A. K. LENTZ, Encéphale **30**:394, 1935.

The results of experiments carried out by Lentz and his collaborators since 1926 are reported in this paper. Saliva was collected by Lashley's suction funnel and measured by Heniké's pressure tube. The subject was placed in a cabin in which he was isolated from all foreign stimuli. The absolute nutritive stimulus, chocolate, was presented on a dish introduced noiselessly into the cabin. The conditioning stimuli—bell, loud noise, metronome and different colored lights—operated within the cabin. The subject was instructed to keep a succinct diary in which he designated by + the stimuli which he found reenforced the response and by — all others. He was to note all reflections concerning the experiment and all self-observations which seemed significant to him. The subject was permitted to read but not to converse during the experiments.

A peculiarity which distinguishes man from the dog is the secretion of saliva in the absence of experimental stimulation—the inevitable secretion. Its amount averages one-half drop in thirty seconds. The effect of a stimulus was expressed quantitatively by the difference between this average and the secretion following the stimulus in question. The quantity was given a positive sign when the secretion exceeded the inevitable amount and a negative sign when it was less than this. The negative quantity represents inhibition. The characteristics of a subject's reactions at a given time were resumed in a formula called the stereotype, consisting of a list of the stimuli, with the sign of each.

In normal human subjects (mostly Lentz' collaborators) conditioned reflexes could be readily formed and were of long duration—two and one-half years after complete interruption of the experiments in one subject. External inhibition was slight. The process of extinction was rapid and followed the same curve as in the dog. However, normal human subjects presented great variability. Some were incapable of forming stable conditioned reflexes, even though they were interested in the experiments. Among these was Lentz himself.

In general the subject's estimate of the sign of the stimulus was fully in agreement with the secretory effect. When the signs of all the stimuli were suddenly and unexpectedly reversed, i. e., the stimuli which were previously presented with the chocolate (reenforced stimuli) were then presented alone, there resulted a rapid understanding of the new situation, as seen in the subject's notes, while the conditioned reflexes took a much longer time to change and did so by a series of oscillations. At first there were a few indecisions and errors in the estimate of the new stimuli. Finally, complete harmony between the conscious estimate and the secretory effect was reestablished. Thus, there was partial dissociation between consciousness and the conditioned reflexes. "To the plasticity of the psyche is opposed the inertia of the conditioned reflexes." The psyche, however, exerted a certain influence on the speed of elaboration and the stability of the reflexes. It is erroneous to suppose with Küppers that conditioned reflexes result from a preestablished notion of the constant result connected with a stimulus. Lentz had previously fallen into the opposite error of reducing, with Watson and some workers of Pavlov's school, all psychic processes to the laws of conditioned reflexes. From the data reported in this paper it is now concluded that "consciousness

represents cerebral and, above all, cortical activity of a particularly superior organization subject to special laws." The salivary conditioned reflexes, while partially mediated by the cortex, represent a more primitive and, to a certain degree, an autonomous activity. "To conceive of a conditioning stimulus and to produce the corresponding reflex are not the same thing."

Lentz' collaborators studied conditioned reflexes of psychopathic patients. In patients with epilepsy associated with dementia Smirnow found that the first reflex was elaborated with normal rapidity but that the reflexes were very unstable. There was no acceleration of the second, third and other conditioned reflexes. Differentiations were difficult to obtain. Immediately after an epileptic seizure the reflexes disappeared and reappeared only after three hours. At times, in the course of an experiment, catalepsy occurred. The presence of salivary secretion could then be observed in spite of complete immobility.

In dementia paralytica Raskina found the conditioned reflexes very unstable. Even the coarsest differentiations could not be elaborated, thus confirming Lentz' previous finding that it is chiefly the processes of inhibition that are paralyzed in these patients. The inevitable secretion increased with general excitement, while the conditioned reflexes disappeared.

Similar results were observed in late stages of schizophrenia by Guerchenovitch, while in the early stages of the disease the conditioned reflexes were normal. Auditory hallucinations interfered with the elaboration of reflexes in response to sound.

In subjects with oligophrenia, Ségal and Sloutskaga found the difficulty of elaborating conditioned reflexes proportional to the degree of mental deficiency. In idiots experimental conditioned reflexes could not be obtained, while the natural conditioned reflexes were present.

Lobatch succeeded in producing conditioned leukocytosis to the sight of food. In subjects with oligophrenia and dementia paralytica in association with nutritive overexcitability, conditioned leukocytosis was very marked.

LIBER, New York.

RELATIONSHIP BETWEEN EXPERIMENTAL LESIONS OF THE LIVER AND BEHAVIOR OF THE ALCOHOLEMIC CURVE. E. BROGGI, Rassegna di studi psichiat. **24**: 579 (July-Aug.) 1935.

Broggi was interested in the relationship of lesions of the liver in chronic alcoholism to the amount of alcohol in the blood stream. He therefore investigated experimentally the presence of alcohol in the blood in normal animals and in animals in which lesions of the liver were produced by experimental intoxication with chloroform. As a result of his experiments Broggi concludes that alcoholemia in dogs with lesions of the liver has the same characteristics as alcoholemia in human beings with chronic alcoholism. He believes that the condition of the liver is an important factor in establishing the type of alcoholic curve of the blood, as alcohol is present for a longer time in experimental animals or in persons in whom lesions of the liver are present. He believes that the alcoholemic curve may be used as an index of liver function.

FERRARO, New York.

FOOT CLONUS. G. G. NOTO, Rassegna di studi psichiat. **24**: 602 (July-Aug.) 1935.

Noto investigated foot clonus in eleven cases by myographic and electromyographic studies. On the basis of graphic studies obtained by the clonograph of Levi in five cases of cerebral lesion, five of medullary lesion and one of extrapyramidal lesion, he confirms the characteristics of organic clonus, consisting in the regularity and limited frequency of the movements (6 oscillations per second). He thinks that the clonus following a cerebral lesion does not differ from that following a lesion of the spinal cord. He, therefore, has been unable to establish differences mentioned by other authors between the medullary and the cerebral type of clonus. He confirms the inhibitory influence of the reflexes

of medullary automatism on the phenomenon of clonus and points out that the Jendrassik maneuver does not inhibit the clonus of spinal cord origin. He insists on the existence of a clonus of extrapyramidal origin. He also mentions that clonus may occur in association with lesion of the peripheral nerves and of the muscular system and that it may also occur in normal persons. He emphasizes the difficulty which at times exists in differentiating the so-called organic from the so-called functional type of clonus.

FERRARO, New York.

TISSUE OXIDATION IN B_1 AVITAMINOSIS AND INANITION. HÅKAN RYDIN, Upsala läkaref. förh. **41**:1 (Oct.) 1935.

This is experimental work which attempts to settle the prevailing controversy concerning tissue oxidation in association with B_1 avitaminosis. Rydin thinks that the diversity of opinions is due probably to the fact that insufficient material has been studied, that no uniformity of methods has been used by various investigators and that most workers have drawn unwarranted generalizations from their results. While some authors obtained reduction in tissue oxidation in cases of vitamin B_1 deficiency others could not substantiate these findings. The controversy is particularly outspoken on the question whether the reduction in tissue oxidation is specific for this type of avitaminosis or whether it is caused by simultaneously occurring inanition. Rydin thinks that the problem may be clarified by the determination of tissue oxidation in association with vitamin B_1 deficiency in various organs of various species, using a number of methods. The author investigated oxidation in muscle tissue in pigeons kept in a state of vitamin B_1 deficiency and inanition under various experimental conditions. Similar determinations under identical experimental conditions were made also with other organs, such as the brain, kidney, liver and blood corpuscles. Tissue oxidation was determined in the same animal with both the indicator and the gaseous analytic method.

Tissue oxidation was studied on finely minced organs with the microrespirometer method of Warburg and Barcroft and with the methylthionine chloride method of Thunberg. Avitaminosis was produced by a diet of polished rice.

A comparison of the muscle oxidation in normal pigeons and in pigeons with vitamin B_1 deficiency showed reduction in the tissue oxidation in the latter group, with or without the use of lactate in the suspension fluid. The tissue oxidation of the normal pigeons used as controls showed great individual variations.

Oxygen consumption of musculature deficient in vitamin B_1 was lower than that of normal musculature, with beriberi broth used in the suspension fluid in both series of tests. Full-grown musculature showed greater tissue oxidation in normal broth than in beriberi broth. Muscle oxidation was lower during the period of vitamin B_1 deficiency. In acute stages of beriberi a greater tissue oxidation was shown than in the chronic stages when the microrespirometer method was used. The methylthionine chloride method failed to give similar results.

Inanition caused by insufficient intake of a diet containing vitamin B_1, with loss of weight comparable to that in pigeons with vitamin deficiency, resulted in lower muscle oxidation. Comparative studies of muscle oxidation in normal, in vitamin-deficient and in starving pigeons revealed similar results in the vitamin-deficient and starved animals. In these two groups the tissue oxidation was decreased as compared with that in normal control pigeons. Determinations with the microrespirometer revealed weaker tissue oxidation in vitamin-deficient brain substance than in the brain tissue of normal and starving pigeons. Vitamin-deficient tissue showed a decreased ability to oxidize lactates. Crystalline vitamin B_1 increases tissue oxidation in the test tube when brain tissue of animals with vitamin deficiency and signs of peripheral neuritis is used. Rydin believes that his experiments with brain substance prove that vitamin B_1 possesses a specific action in the oxidation of brain tissue.

NOTKIN, Poughkeepsie, N. Y.

Neuropathology

FULMINATING HEMORRHAGIC ENCEPHALITIS. A. LEVINSON and OTTO SAPHIR, Am. J. M. Sc. **190**:42 (July) 1935.

The clinical histories and autopsy observations in five cases of fulminating encephalitis are recorded. Death occurred suddenly in one instance and unexpectedly within from four and one-half to forty-eight hours after the onset in the other cases. The outstanding symptom was respiratory difficulty. There was no uniformity in cerebrospinal fluid findings, which depend on degree of meningeal involvement. Bronchopneumonia was present in four cases in which the lungs were examined. Extravasation of red blood corpuscles in addition to infiltration with lymphocytes and a few neutrophils occurred in three brains. In the other two cases, a larger number of neutrophils was present, and red blood cells and lymphocytes were also found free in the tissue. The marked hyperplasia of the lymphadenoid structures in three patients raises the question of the presence of status thymolymphaticus and the part which this played in the suddenness of the onset and death. It is suggested that the suddenness of symptoms and death might be due to the presence of a generally lowered resistance as reflected in enlargement of the lymphadenoid structures.

MICHAELS, Boston.

SUDDEN DEATH DUE TO HEMORRHAGE INTO SILENT CEREBRAL GLIOMAS. OSCAR T. SCHULTZ, Am. J. Surg. **30**:148, 1935.

A previous study revealed that from 49 to 59 per cent of all deaths reported to three medical examiners' offices and two coroners' offices were due to natural causes. It is usually the suddenness of such deaths that makes their investigation necessary. Cardiovascular disease, including hemorrhage, is by far the most frequent cause of sudden death. The most common form of fatal hemorrhage is that which occurs in the brain as a result of vascular disease. A rare form of intracranial hemorrhage is that which results from disruption of the vessels of a previously unrecognized tumor of the brain. The clinical signs and symptoms are those caused by the hemorrhage. Death may occur within a few hours after the onset of such symptoms. Three cases of fatal hemorrhage into a silent glioblastoma of the brain are described. With the sudden onset of symptoms of intracranial hemorrhage, the relative youth of the patient as compared with the age of those in whom apoplexy most often occurs may help in reaching the correct antemortem diagnosis of hemorrhage into a tumor of the brain.

FROM THE AUTHOR'S SUMMARY. [ARCH. PATH.]

THE TRANSMISSION OF EQUINE ENCEPHALOMYELITIS VIRUS BY AEDES AEGYPTI. M. H. MERRILL and C. TENBROECK, J. Exper. Med. **62**:687, 1935.

In confirming Kelser's work on the transmission of equine encephalomyelitis of the western types by Aedes Aegypti it has been learned that the mosquitoes must be fed virus of high titer if positive results are to be secured. A period of from four to five days after feeding either on infected guinea-pigs or on brain containing virus must elapse before the disease is transmitted by biting, but after this time transmission regularly results for a period of about two months. By inoculation virus can be demonstrated in the bodies of infected mosquitoes for the duration of life. Although virus multiplies in the mosquitoes and is generally distributed in their bodies, repeated attempts to demonstrate it in the eggs of females known to be infected as well as in larvae, pupae and adults from such eggs have been uniformly without result. Larvae have not taken up virus added to the water in which they live. Male mosquitoes have been infected with virus by feeding, but they have not transmitted the virus to normal females, nor have males transmitted the virus from infected to normal females. When virus of the eastern instead of the western type is used, transmission experiments with Aedes Aegypti result

negatively. Apparently this virus is incapable of penetrating the intestinal mucosa of the mosquito. If, however, it is inoculated into the cavity of the body by puncture with a needle it persists, and transmission experiments result positively.

FROM THE AUTHORS' SUMMARY. [ARCH. PATH.]

LIPOMA AND OSTEOLIPOMA OF THE BRAIN. S. J. SPERLING and B. J. ALPERS, J. Nerv. & Ment. Dis. **83**:13 (Jan.) 1936.

Lipoma is one of the rare types of tumor of the brain. There is no characteristic symptomatology, the diagnosis in most cases having been made at autopsy. It is situated chiefly at the base of the brain, around the optic chiasm, tuber cinereum and posterior perforated spaces. Histologically lipoma of the brain is similar to lipoma elsewhere. Much speculation has arisen as to the origin, and Sperling and Alpers review the various theories. Recent opinions seem to favor the view that the tumors originate from undifferentiated mesenchymal rests in the meninges.

HART, Greenwich, Conn.

ACTION OF ROENTGEN RAYS ON SYRINGOMYELIA. J. HEINISMANN and S. SAVENKO, Encéphale **30**:562, 1935.

The first case of syringomyelia with improvement by roentgen therapy was reported in 1905 by Raymond, Oberthur and Delherm. Many similar reports have since been published. The mode of action of roentgen rays can be explained in three ways: (1) by action on the proliferated neuroglial tissue; (2) by action on young and radiosensitive blood vessels, and (3) by action on the hydromyelia, when present, similar to that of roentgen rays on hydrocephalus. Only two cases of syringomyelia in which treatment with roentgen radiation was given have been reported with autopsy. In one case, in which the clinical syndrome was that of syringomyelia, the condition pathologically was hydromyelia. In both cases the gliosis about the cavity was scant in the regions which had been irradiated, while it was abundant elsewhere.

A third case, with autopsy, is reported. The clinical signs indicated syringomyelia. Improvement was slight after irradiation. Autopsy showed a hydromyelic cavity extending from the lower part of the medulla to the lower portion of the lumbar region of the cord. The cervical portion of the cord was reduced by the cavity to a thin band. In the cervical region the ventral two thirds and part of the dorsal third of the cavity were lined by ependyma. The remainder of its circumference was lined by collagenous tissue. About the cavity was a thin layer of fibrous glia, without cells. A similar appearance was noted in the thoracic portion of the cord. In the lumbar region the cavity was smaller, and its outline was festooned. It was almost surrounded by ependyma, which in places was many layered and penetrated into the subjacent tissue. Outside the ependymal layer was a thick band of fibrous glia, which extended into the right ventral horn. At all levels the gliosis contained numerous vessels with thick, hyalinized intima, forming volutes. The pia was likewise hyalinized. The ganglion cells were flattened and shriveled but did not show changes recalling those produced experimentally by roentgen rays.

The conclusion is reached that the blood vessels and collagenous tissue were not affected by the roentgen rays. That the layer of gliosis was thinner in the irradiated than in the untreated regions is taken as evidence of the inhibitory effect of roentgen rays on glial proliferation. The slight transitory nature of the clinical improvement in this case is attributed to the fact that the cavity was surrounded chiefly by collagenous tissue, and only to a lesser extent by glia.

LIBER, New York.

HEMIATROPHY OF THE CEREBRUM AND CEREBELLUM DUE TO CHRONIC ENCEPHALITIS. BARAHONA FERNANDES, Ztschr. f. d. ges. Neurol. u. Psychiat. **153**:506 (Sept.) 1935.

Fernandes reports the case of a woman who at 26 had a vague cerebral disturbance after delivery. Three years later she began to have epileptic attacks. At 51 the attacks became more frequent, with periods of confusion, depression and hypochondriasis. At 54 the patient had a second attack of right hemiplegia, with convulsions on the right side. At 59 the epileptic seizures became more frequent. In the interparoxysmal periods she was restless, euphoric and dysarthric; she perseverated, had difficulty in finding words and was apraxic. In addition, she presented a picture of organic dementia. She died in status epilepticus.

Autopsy showed verrucous endocarditis, with patent foramen ovale. The left cerebral and cerebellar hemispheres were definitely atrophic, especially the temporal lobe. Microscopically the cerebrum showed a diffuse loss of ganglion cells and myelin sheaths, with gliosis, which was most marked in the temporal lobe. There was definite round cell infiltration with lymphocytes and plasma cells, especially in field 37. There were typical ischemic areas in the cornu ammonis, such as are usually encountered in epilepsy of long standing. There were also scattered old areas of softening, probably due to emboli. The changes were all much less marked on the right. The inflammatory changes on the right were less marked, though old areas of softening were more numerous.

Fernandes states that this was a case of encephalitis, with the probable period of invasion in the twenty-sixth year. This infection persisted to the end. The emboli probably originated from the endocarditis. The emboli alone were not sufficient to account for the marked atrophy of the brain. The unusual features in the case were the long duration (thirty-three years) and the limiting of the pathologic process to one side. Fernandes also notes that the changes in the cornu ammonis were more marked on the left side. He suggests that vasospasms may have played a rôle in spreading infectious material throughout the brain. He comments on the sparing of the first temporal convolution and calls attention to the frequency of embolic infarcts in the olive, which were present also in this case.

While atrophy of the cerebellum has been reported following infectious disease in children, it has not been reported in the adult up to the present. The unilateral degeneration following infectious disease is distinctly unusual. There was only slight evidence of inflammation in the cerebellum. The occurrence of embolism, infection and changes in the tissue secondary to convulsions probably all played a part in the atrophy of the cerebellum. The neocerebellar tissues were much more profoundly affected than the paleocerebellar structures. Unilateral atrophy of the cerebellum is not seen in association with senile or presenile psychosis.

SAVITSKY, New York.

FURTHER EXPERIMENTAL STUDIES ON THE EFFECT OF INSULIN ON THE BRAIN. A. STIEF and L. TOKAY, Ztschr. f. d. ges. Neurol. u. Psychiat. **153**:561 (Sept.) 1935.

Stief and Tokay studied the effect of subcutaneous, cisternal and intracerebral injections of insulin on the brains of sixteen dogs. There were definite variations of susceptibility to insulin in the different animals. Small doses did not produce significant histologic changes. The mild clinical changes and perhaps the anatomic alterations were apparently reversible. No difference was found in the reactions of the animals to cisternal as compared with subcutaneous injections. The severity of the anatomic and clinical changes was proportional to the amount of insulin injected. There was no evidence of direct injury to the neural parenchyma. The histologic changes were unquestionably due to circulatory insufficiency to circumscribed parts of the brain. The occasional round cell infiltration in the parenchyma was considered to be the result of prestasis. The meningeal reaction in the cases in which cisternal injection was given was probably a direct response to the

insulin. The cerebral cortex was most seriously involved, the fewest changes occurring in the occipital lobes. In the cerebellum the Purkinje cells were apparently the most vulnerable of the neural elements.

The effect of cisternal injection was usually delayed because of the time required for absorption into the meningeal vessels. The cerebral circulation was not affected directly by insulin but responded secondarily to hypoglycemic shock, after resorption of insulin into the veins from the subarachnoid spaces. Even in cases in which insulin was injected into the brain itself no changes were noted in the parenchyma or vessels at the site of injection. Histologic alterations were noted mainly at a distance from the part of the brain into which injection was made. It was noted also that insulin can penetrate the hemato-encephalic barrier and pass directly into the blood stream.

SAVITSKY, New York.

HISTOPATHOLOGIC CHANGES IN THE BRAIN IN MANGANESE POISONING. H. STADLER, Ztschr. f. d. ges. Neurol. u. Psychiat. **154**:62 (Oct.) 1935.

The brain of a man aged 56 was studied. He had worked from 1910 to 1915 and again from 1918 to 1921 with manganese. Soon after the onset of severe backache at 46, in 1922, there developed a clinical picture typical of manganese poisoning—parkinsonism with hypomimia, seborrhea, flexion of the trunk, micropraxia, micrographia and palilalia. Six months before death arterial hypertension developed. The patient died, with cardiac decompensation. Autopsy revealed softening in the wall of the left ventricle of the heart. The heart was enlarged with hypertrophy of the left ventricle and definite sclerosis of the coronary arteries.

There were multiple discrete areas in the brain in which ganglion cells had disappeared and had been replaced by glial tissue. The areas varied in size; none was very large. The glia showed little proliferation of fibers. In the center of each area there was usually a precapillary vessel, with no change in its wall. Around the areas were gitter cells. The most marked changes were present in the striatum and pallidum. The cerebral cortex was also involved, especially in the parietal and frontal regions. The changes were milder in the thalamus, hypothalamus and cerebellum. Here and there fresher foci, due to more recent ischemia, were noted. In all instances the tissue changes were around blood vessels which were not themselves altered. In the pallidum, however, such a constant relation of the tissue changes to the blood vessels was not observed. There were diffuse alterations in the ganglion cells, with replacement by glial tissue. No pigment was noted in the glia cells. These cell changes were most intense in the inner part of the pallidum. The white matter was practically spared. There was slight involvement of the pallidofugal fibers, which was probably secondary to the parenchymatous changes noted. The substantia nigra was normal. No manganese was demonstrable by chemical analysis of the brain substance.

Arteriosclerosis and hypertension cannot be considered to be the causes of the changes in the tissues of the brain. The hypertension was noted only six months before death, while the neurologic signs had been present for about a decade. The cerebral vessels were not arteriosclerotic. The probable explanation for these anatomic changes in the brain is functional circulatory disturbances, such as were described by Ricker and Spielmeyer. These were in the nature either of spasms or stasis in blood vessels. The elective action of manganese on the pallidal cells may perhaps be explained on the basis of a special affinity of manganese for iron, which is present especially in this part of the brain.

SAVITSKY, New York.

NEUROPATHOLOGIC CHANGES IN A CASE OF HEMORRHAGIC ZOSTER WITH MYELOPATHY, PERNICIOUS ANEMIA AND GASTRIC CANCER. LÁRUS EINARSON, Laeknabladid **20**:113 (Dec.) 1934.

Einarson reports the case of a woman aged 58, who suffered for a number of years from pernicious anemia. Toward the end of life there developed a gastric

cancer and eighteen days before death hemorrhagic herpes zoster, with peripheral sensory distribution in the region of the lumbo-inguinal and lumbofemoral segments. A detailed examination of the spinal cord and all the spinal ganglia was made. The cord showed changes in the posterior columns, lateral and spinocerebellar tracts and Clarke's column. Of particular interest were the changes in the spinal ganglia, which consisted of chromatolytic changes of the cells, with more or less pronounced lipoid degeneration, pigment atrophy and secondary vacuolation—changes which are described under the name of frothy degeneration. The deposit of fat was in reverse ratio to the number of Nissl bodies, which Einarson considers to be an expression of disturbance in cellular metabolism, especially a sign of decreased internal respiration. The nuclear changes were in proportion to the vacuolation of the protoplasm. The spinal ganglia from the eleventh thoracic to the second lumbar inclusive showed the presence of carcinomatous metastases. No normal nerve cells were observed in these ganglia. In addition to chronic alterations, the cells also showed evidence of acute changes, such as toxic vacuolation, coagulation necrosis and neuronophagia.

Einarson is in favor of the theory that in combined sclerosis there is first involvement of the cells in the spinal ganglia, with secondary degeneration of the tracts. He also supports the hypothesis that the early clinical manifestations, especially acroparesthesia, are due to the cellular changes in the spinal ganglia.

NOTKIN, Poughkeepsie, N. Y.

Psychiatry and Psychopathology

THE SCOPE OF THE PROBLEM OF DELINQUENCY AND CRIME AND THE CONTRIBUTIONS OF PSYCHIATRY. GEORGE M. LOTT, J. Crim. Law & Criminol. **26**:61, 1935.

This article reports a study of 407 cases referred to the Rhode Island State Public Welfare Commission's Psychiatric Clinic. The subjects were of both sexes and of all ages and conditions, from dependent and delinquent children to repeated offenders and "long termers." Forty-one and one-half per cent of the patients were juveniles, and only 13.5 per cent of those examined were from the community, the rest being in institutions. Nine per cent had serious physical disabilities, and it was found that insecurity and feelings of inadequacy, rejection and difference were prominent. Thirty-four per cent could be sent back into the community. Thirty-five per cent needed prolonged institutional or other treatment, and 14 per cent were abnormal but did not suffer from psychosis. Two thirds of these were definitely mentally defective. Lott presents a prognostic classification into four groups: (1) persons who can be readily adjusted in or returned to the community—probational material; (2) persons requiring institutional or other treatment, with a doubtful prognosis; (3) persons mentally or emotionally abnormal within the limits of sanity and (4) persons requiring care in a hospital for mental disease. From the point of view of prognosis the predominant number of adults were in the group of persons with a doubtful prognosis, while over three fourths of the juveniles were in group 1, or the group with a good prognosis. The intelligence of the subjects in the group was tabulated and it was found that persons with an intelligence quotient of 100 or over and persons intermediate between those with mental deficiency and those in the group with an intelligence quotient of 100 predominated in the group with a good prognosis, while the mentally deficient patients were to a great extent in the group with a doubtful prognosis or emotional abnormality. The prognosis in the case of adults tended to be less satisfactory than that in the same groups of juveniles. Forty-two per cent of the cases were in the group of persons with intermediate intelligence. Lott points out that psychiatry can contribute a method of approach to the cure of a concealed personality twist or other cause of unacceptable behavior. Economic adjustment of persons released from institutions

can be the function of a clinic, and the clinic may aid in the selection of material for probation and parole. Thirty-four per cent of the patients presenting the worst problems in one state could be fitted back into the community. Lott urges further use of psychiatric clinics and the collection of data from clinics assigned to juvenile and other courts, from probation, parole and children's bureaus, juvenile and adult corrective schools, jails and prisons to deal with the problem of crime.

SELLING, Detroit.

PREDICTION METHODS APPLIED TO PROBLEMS OF CLASSIFICATION WITHIN INSTITUTIONS. GEORGE B. VOLD, J. Crim. Law & Criminol. **26**:202, 1935.

Burgess and the Gluecks have each developed a method of parole prediction. Burgess' method is to inspect the percentage distribution in relation to violators or nonviolators of parole. Twenty-one factors were selected as important, and tables were made. The Gluecks utilized the coefficient of mean square contingency and constructed tables on the basis of a smaller group of factors—from six to thirteen. Vold finds that there is little difference in results with the two methods. A group of 282 cases in the Minnesota State Prison gave a correlation of about 0.5 between the predicted outcome on parole and the estimate of a parole officer. The correlation for predicted outcome and actual outcome was 0.4, and the estimate of the parole agent correlated against actual outcome, 0.3. Vold devised a technic to be used in classifying inmates, which correlates well with both the Burgess and the Glueck technic. He has the prisoner rated by as many as possible of the officers of the institution who come in contact with him; they are asked to rate him as being among the best one-fourth or the worst one-fourth or as belonging in the middle half. This information is then checked against each record, after which the usual technic, similar to parole prediction procedures, is followed. By these means, the conduct of prisoners was found to be predictable.

SELLING, Detroit.

PERSONALITY STUDIES IN MIGRAINE. OLGA KNOPF, J. Nerv. & Ment. Dis. **82**: 270 (Sept.); 400 (Oct.) 1935.

Crookshank classified patients with migraine as thinkers rather than doers and regarded the symptom as a defense, flight or excuse mechanism. Touraine and Draper made a study of fifty cases, with similar results.

Thirty cases of migraine are reported in this study. Practically none of the twenty-two women in the group had a well balanced conception of woman's rôle, and four were not prepared for menstruation. The onset of the symptom in nine of the twenty-two women was within a year of the menarche. Most of the patients came from large families. On the whole, persons with migraine are of the "goody, goody" type. They are ambitious, reserved, repressed, dignified, sensitive, domineering and resentful and possess little sense of humor. The genital phase of the adjustment of all the twenty-two women was incomplete. The records for the thirty patients are summarized. The conclusion is that the psychologic readjustment of the patient should be given a greater place in the therapy of the patient with migraine than it has had thus far.

HART, Greenwich, Conn.

ON THE CAUSATION OF MENTAL SYMPTOMS. MAX LEVIN, J. Ment. Sc. **82**:1 (Jan.) 1936.

Levin points out that the famous observation by Goltz on the dog can be applied to patients with schizophrenia. He found that a dog's paw is not permanently paralyzed as an organ of locomotion by destruction of the cortex but remains permanently paralyzed for all the actions in which it is employed as a hand. This observation can be applied qualitatively but not quantitatively to patients with schizophrenia. In the dog the use of the paw as a hand is completely lost. In

most patients with schizophrenia the use of the thinking apparatus as a complex instrument is not completely, but only partially, lost. The schizophrenic patient in whom the disease is only moderately advanced may be able to think abstractly only to a moderate degree. In principle, however, the law revealed by Goltz in his observations on dogs applies in schizophrenia as well as in all diseases of the higher centers.

In the dog the nervous mechanism governing the paw may be said to consist of two levels: a lower level which subserves the more automatic functions of the paw, such as those in locomotion, and a higher level, which subserves the more special functions, such as those in which the paw is used as a hand. Destruction of the higher level results in loss of more special functions, more automatic functions being retained. Except for one circumstance, this is an exact analogy to the cerebral disturbance which results in schizophrenic thinking. The substrate of mentation—highest cerebral centers—may, according to Jackson, be assumed to consist of an indeterminate number of layers. (These, of course, are physiologic and not morphologic layers.) Lower layers form the substrate of a primitive kind of mentation, higher layers, of a more advanced (a more special) kind of mentation. When the highest layers are paralyzed the thinking apparatus is reduced to a more primitive type of organization. In children and in savages the highest layers have not yet developed; in civilized adults with schizophrenia they have attained some degree of development but have subsequently been reduced by disease to a lower level.

KASANIN, Chicago.

AN ATTEMPT AT A PHYSIOLOGIC INTERPRETATION OF PARANOIA AND OBSESSIONAL NEUROSIS. J. P. PAVLOV, Encéphale **30**:381, 1935.

When a conditioned reflex is formed, the first reaction of the animal is a movement toward the conditioned stimulus. The animal turns toward it and tries to come in contact with it. In one dog such an approach reaction toward the source of a sound took place in response to any conditioned stimulus and persisted a year and a half after cessation of the conditioned stimulus which had originally elicited the reaction. At each stimulation the dog turned toward the source of the original sound stimulus and ceased to do so only when a bowl of food approached its mouth. This reaction must be considered pathologic, for it was in "flagrant contradiction to the real relations, i. e., had no sense." Following Pavlov's usual practice in cases of experimental neurosis in dogs, bromide was given. The pathologic reaction then disappeared in response to all but the original conditioned stimulus. Of other similar cases observed later, "radical cure" was obtained in one by bromide treatment. In regard to this Pavlov stated: "It is clear that we have before us a particular disturbance in the functioning of the nerve cells, namely, a pathologic change in the relations between the two components of their activity, the processes of excitation and inhibition. The former predominates. Bromides, by reenforcing inhibition, have reestablished the normal relation." This disturbance in the excitation-inhibition balance is named "pathologic inertia." It can be explained either by increased stability of excitation or by weakened inhibition. The action of bromides and the fact that this disturbance occurred particularly in castrated animals support the latter hypothesis. One of the principal effects of castration is the weakening of inhibition.

All these observations confirm Pavlov's long established contention that "it is perfectly possible to obtain a strictly limited pathologic point in the cortex by physiologic processes, i. e., without any mechanical action."

Certain conditions in man, such as stereotypy, iteration, perseveration, obsessive neurosis and paranoia, can be explained by pathologic inertia. Two sets of factors, predisposing and determining, are concerned in producing these states. A predisposition can be innate, analogous to the "nervously weak" and the "nervously strong but unbalanced" dogs, who are particularly susceptible to experimental neuroses, or else it can be acquired by the sorrows and misfortunes of life, psychic shocks and traumas, infections and intoxications, just as in the dog it can be

acquired by castration. The determining causes in the experimental animals are of two orders: overexcitation and the violent collision of two contrary processes. The essential difference between obsession and paranoia, namely, the presence or absence of self-criticism, is explained by a difference in the intensity of the pathologic inertia. When the inertia is sufficiently intense, it is "refractory to the influences of the healthy regions of the cortex, which explains the lack of criticism." A further factor in some cases is the "ultraparadoxal phase," which is best illustrated by an example. In an animal the cortex of which is in a state of inhibition, either generalized or localized to "the region of action of metronomes," a metronome of a frequency which had previously been "positive" becomes inhibitory and a "negative" becomes excitatory.

In conclusion, Pavlov declares that "hereafter neurologists and psychiatrists will have to consider three well established physiopathologic facts: (1) the possibility of the existence in the cortex of sharply isolated pathologic points, (2) pathologic inertia of the process of excitation and (3) the ultraparadoxal phase."

LIBER, New York.

A CONTEMPORARY MENTAL EPIDEMIC: THE BELGIAN APPARITIONS. AUGUST LADON, Hyg. ment. **30**:205 (Dec.) 1935; **31**:1 (Jan.) 1936.

This is a detailed account of the phenomena occurring in Belgium from November 1932 to April 1935, in which a large number of persons saw the miraculous representation of the Virgin Mary. First, at Beauraing, on Nov. 29, 1932, several children saw what they interpreted as the Holy Virgin in a garden adjacent to a religious institution. On successive days more and more persons came to gaze on this modern miracle. Forty-seven made formal declaration of these facts. These observers ranged in age from 5 years to past 60 and represented all ranges of intellectual capacity. Still others swore to a similar phenomenon at Onkerzeele. The details observed varied widely—sometimes only a vague light was seen, at other times a clearly outlined figure of the Virgin.

In analyzing the situation from a psychiatric angle the phenomena are looked on as illusions and hallucinations occurring in a population of general good sense and uprightness. The disarrangement in life following the war and incident to the world depression, the aspirations to a higher spiritual level of equilibrium, a sense of the inversion of values so out of keeping with these aspirations—these provided the background for a veritable psychic epidemic. The great value of this account lies not so much in the interpretation as in the authentic presentation of observations of a most interesting phenomenon.

ANDERSON, Los Angeles.

THE GALVANIC CUTANEOUS REFLEX IN ENDOGENOUS DEPRESSIONS. H. BÜSSOW and K. W. ESSEN, Monatschr. f. Psychiat. u. Neurol. **90**:326 (March) 1935.

The galvanic cutaneous reflex, or the so-called psychogalvanic reflex, is a manifestation of activity of the autonomic nervous system and can be elicited not only by emotional stimuli but by acts such as coughing, sneezing or deep breathing, which may be completely independent of the psyche. In the experiments of Büssow and Essen, coughing was employed to elicit the reflex. With this procedure curves are obtained which can be quantitatively evaluated and used as an indicator of the readiness of the autonomic nervous system to react. The features characteristic of normal curves have been described by Essen in a previous investigation on healthy persons. Forty-one patients who were suffering from endogenous depressions were studied by Büssow and Essen. Abnormal curves were obtained from thirty-two patients. The changes consisted of one or more of the following features: increase in the latent period, prolongation of the duration of the reaction, wavelike irregularities in the curve and rapid exhaustibility of the reaction on repeated tests. The reflex was normal in four of thirty-

four retarded patients and in five of seven agitated patients. While the changes recorded are by no means specific for depressive conditions, they reveal the presence of a somatic anomaly that occurs with greater regularity than most of the somatic alterations hitherto described in such disorders. The observations provide direct evidence of abnormality of function of the autonomic nervous system in endogenous depressions

ROTHSCHILD, Foxborough, Mass.

Diseases of the Brain

HYPERGLYCEMIA AND PARESIS: REPORT OF TWO CASES. L. MINOR BLACKFORD and JOHN H. VENABLE, New England J. Med. **214**:140 (Jan. 23) 1936.

Clinical reports are submitted of two cases of dementia paralytica in which hyperglycemia was noted. References are given which show that disturbance of the base of the brain can cause increase in the amount of blood sugar. It may be assumed that late syphilis of the central nervous system can affect these areas.

MOORE, Boston.

INDUCED WATER RETENTION IN DIAGNOSIS OF IDIOPATHIC EPILEPSY. J. L. CLEGG and F. T. THORPE, Lancet **1**:1381 (June 15) 1935.

Clegg and Thorpe state that after the exclusion of other causes of seizures there remain cases in which a differential diagnosis between idiopathic epilepsy and hysteria can be made only when a seizure has been witnessed by a trained observer. This is often difficult when the attacks are infrequent, even though the patient is detained in the hospital for this purpose. In this study the authors used the technic described by Jacobsen to induce a positive water balance. Large quantities of water are administered by mouth, accompanied by hypodermic injections of pitressin. An increase in body weight of from 3 to 6 per cent is taken as an indication that a positive water balance has been produced. The test was conducted on sixteen adults with epilepsy and on seven persons as controls who did not have epilepsy. Of the sixteen patients with epilepsy, twelve had one or more convulsions during the administration of pitressin in combination with water; in two of the four patients in whom no seizure occurred the injections had to be discontinued because of hiccup and vomiting, and in the other two the convulsions were normally very infrequent. In none of the seven persons used as controls was a seizure induced by the injection of pitressin and water, nor was it induced in any of the patients with epilepsy to whom water or pitressin was administered alone.

Cryoscopic examinations were made on the blood during the tests. The osmotic pressure of the blood depends on its molecular concentration, which can be accurately determined by the estimation of the freezing point. Any retention of water in the blood stream will be shown by elevation of the freezing point. Samples of blood were taken at intervals throughout the test. In three patients with epilepsy the curve for the freezing point was obtained. This showed a progressive lowering of the osmotic pressure of the blood in proportion to the quantity of water ingested and the increase in body weight. Since water retention is accompanied by marked lowering of the osmotic pressure of the blood serum, Clegg and Thorpe believe it is probable that the resulting exudation of fluid into the nerve tissues is the precipitating factor in the production of the seizures.

WATTS, Washington, D. C.

POST-OPERATIVE INTRACRANIAL THROMBOSIS IN CHILDHOOD. P. R. EVANS, Lancet **2**:12 (July 6) 1935.

Intracranial thrombosis may occur in children as in adults after local operations, such as mastoidectomy. In 1910 Schwenninger stated that classic thrombophlebitis after operation is never seen before puberty—unlike septicemic thrombophlebitis, which attacks infants. Evans believes that Schwenninger's

statement, however, was too sweeping, though he brought out the useful distinction between the septicemic and the nonsepticemic form.

On observing 2 cases of postoperative intracranial thrombosis in children, Evans studied the records of 11,500 postmortem examinations at the Hospital for Sick Children and found 6 other instances. In 4 instances the operation was abdominal (in 2, herniotomy) and in 4, buccal (in 3, for cleft palate). Infection occurred in 1 of the cases of abdominal operation and in 3 cases of buccal operation. The superior longitudinal sinus was involved in 5 instances.

Evans believes that it is difficult to learn why these nonseptic thromboses occur and suggests no cause in 3 of the cases. Untoward signs in these cases were first noted at intervals varying from a few hours to two weeks after the operation. There was no significant difference in this respect between cases in which there was infection and cases in which there was none. Signs and symptoms varied with the position of the lesion. The commonest site was the middle portion of the superior longitudinal sinus, so that venous congestion of the cerebral cortex, especially the rolandic area, occurred early and was followed by thrombosis. Consequently, motor symptoms were frequent. Later, softening of the brain occurred and was often followed by hemorrhage, giving rise to further symptoms. Drowsiness and vomiting were frequent.

WATTS, Washington, D. C.

DIFFERENTIAL DIAGNOSIS OF BASILAR MENINGITIS AND HYPOPHYSEAL TUMOR. I. F. FISCHER, Ann. d'ocul. **172**:789 (Sept.) 1935.

Fischer reports the case of a man aged 62 who presented Argyll Robertson pupils and grayish disks; the visual field was contracted concentrically, especially for red. The Bordet-Wassermann reaction was positive. A diagnosis of beginning tabetic atrophy of the optic nerve was made, and specific treatment was administered. After several weeks the visual fields presented bitemporal hemianopia for red. A roentgenogram of the base of the skull showed enlargement of the sella turcica. Differential diagnosis of hypophyseal tumor and specific basilar meningitis could not be made with certainty. Ventriculography indicated that a hypophyseal tumor was present.

BERENS, New York.

SPONTANEOUS AND LONG-STANDING REMISSIONS IN THE COURSE OF EVOLUTION OF TUMOR OF THE BRAIN. T. DE MARTEL, H. SCHAEFFER and J. GUILLAUME, Presse méd. **44**:762 (May 9) 1936.

The steady progression of symptoms is generally accepted to be a characteristic feature of the evolution of intracranial neoplasm. The authors report three cases of tumor of the posterior fossa, in the evolution of which there occurred remissions of six, twelve and eighteen months, respectively. All symptoms, focal as well as general, including choked disk, regressed rapidly and without any apparent cause, resulting in almost complete recovery. In all three cases the operation performed after a relapse revealed a cystic astrocytoma fibrillare of the cerebellum. Rupture of the wall of the lateral ventricle at its weakest point, that is, in the choroid fissure of Bichat, thus establishing communication between the ventricular system and the subarachnoid space, is one of the mechanisms which the authors demonstrated by means of a ventriculogram, which explains remissions, at least in some cases of tumor of the posterior fossa. The authors cite such a case, in which, after a particularly severe exacerbation of headache, there occurred sudden relief from symptoms, with abrupt cessation of headache and regression of the focal signs of the tumor. A ventriculogram showed a communication between one of the ventricles and the subarachnoid space on the same side, and operation confirmed the clinical findings. An identical mechanism was disclosed by ventriculograms in two other cases in which observation was made.

YAKOVLEV, Waltham, Mass.

NONSUPPURATIVE OTOGENOUS ENCEPHALITIS OF THE TEMPORAL REGION. J. A. BIJLEVELD, Rev. d'oto-neuro-opht. **14**:81 (Feb.) 1936.

Nonsuppurative otogenous encephalitis is almost always diffuse. It is characterized by continuous high fever, without slowing of the pulse, and headache, which is less characteristic than that of cerebral abscess. Bijleveld reports the case of a child aged 5 years, in whom the symptoms were different from those in the classic form of the disease. After bilateral mastoidectomy the fever was of the pyemic type; the pulse was very rapid; cultures of the blood were sterile; meningeal reaction was not present, and there were marked focal signs (mimic facial paralysis on the left side). The right temporal lobe was explored with negative results so far as the presence of pus was concerned, but softened cerebral tissue was aspirated through the exploring needle. Since there was no improvement in the condition of the patient radical mastoidectomy was performed, and the patient made a prompt and complete recovery.

Nonsuppurative otogenous encephalitis is probably more frequent than is suspected. A focus of osteitis somewhere is indispensable for the production of encephalitic infection, and one can never be sure that otitis media is completely cured. Therefore, in a case of otogenous encephalitis or cerebral abscess, if improvement is not prompt after simple mastoidectomy or if, after ample drainage of an abscess, this tends to extend and the patient is not apyretic in a few days, radical mastoidectomy should be done at once. DENNIS, San Diego, Calif.

SPIROCHETES IN THE LYMPH SYSTEM OF PATIENTS WITH DEMENTIA PARALYTICA. J. L. SKOBSKI, Monatschr. f. Psychiat. u. Neurol. **89**:365 (Oct.) 1934.

Skobski attempted to determine whether spirochetes were present in the lymph glands in cases of dementia paralytica. Five patients who had had malarial therapy and had been hospitalized for several years were studied. In each case material obtained by puncture of the inguinal lymph nodes was injected into the testes of a rabbit. In all the animals chronic orchitis developed, which clinically resembled syphilitic inflammation. Spirochetes were observed in smears obtained from three animals, though examination of the involved tissue after it had been fixed in formaldehyde and stained by the Levaditi method failed to disclose any organisms. ROTHSCHILD, Foxborough, Mass.

CONTRIBUTIONS TO THE NEUROLOGIC FEATURES OF THE FRONTAL LOBE AND CORPUS CALLOSUM: A FRONTAL LOBE-CORPUS CALLOSUM SYNDROME IN ASSOCIATION WITH TUMOR. L. HALPERN, Schweiz. Arch. f. Neurol. u. Psychiat. **37**:68 and 253, 1936.

Halpern reports the clinical and pathologic observations in four cases of tumor of the frontal lobe and corpus callosum. In a case of tumor of the right frontal lobe which had invaded the anterior part of the corpus callosum to a slight extent there was miosis of the left pupil, with narrowing of the corresponding palpebral fissure, eventually succeeded by mydriasis, with widening of the fissure. This was attributed to involvement of an area in the frontal lobe concerned with the central regulation of sympathetic activity. Signs of impaired pyramidal function and severe disturbance of equilibrium on the left side of the body, as well as increased irritability to rotation of the left labyrinth, were recorded. Symptoms regarded as unequivocally those of localization in the frontal lobe were akinesia and mental disturbances characterized chiefly by inability to distinguish figures from the background.

Signs of a general increase in pressure in the second case were limited largely to papilledema, which was more marked on the right side. Since the patient was subject not only to jacksonian seizures on the left side, on which involvement of the pyramidal tracts could be demonstrated, but to tonic spasms, with turning

of the body to the right, it was evident that the lesion was bilateral. Further evidence of localization in the frontal lobe consisted in mental symptoms and severe astasia, whereas involvement of the corpus callosum was betrayed by difficulty in control of the bladder and apraxia of the left hand. Rotation provoked a tonic spasm having the characteristics of a neck reflex. At autopsy a glioma of the right frontal lobe and corpus callosum and an isolated tumor of the left frontal lobe were observed.

In the third case a cylindric tumor was observed in the medial portion of the left frontal lobe immediately above the corpus callosum, which was also invaded. A severe mental disturbance which suggested schizophrenia was a feature in this case. Signs of increased intracranial pressure were lacking.

In a case of tumor invading the anterior portions of both hemispheres, including the corpus callosum, involvement of the left frontal cortex immediately anterior to the motor area was indicated by jacksonian seizures, reflex changes and a slight flexion contracture of the right arm, as well as by limitation of conjugate deviation of the eyes to the right. Involvement of the right frontal lobe was evidenced by astasia, with a tendency to fall to the left and hyperirritability of the left labyrinth to caloric stimulation. Apraxia of the bladder and of the left hand was ascribed to invasion of the corpus callosum by the tumor. There was a terminal psychosis of the Korsakoff type.

Halpern expresses the view, contrary to the opinion frequently stated, that although mental disturbances are frequent in association with tumor involving the frontal lobe and corpus callosum, a lesion of the latter structure has nothing to do per se with such symptoms and, further, that the various mental changes encountered in disease of the frontal lobe can all be traced to a fundamental inability to grasp the essential features of a situation. The patient is indifferent because to him everything is of equal value. Although there is scarcely a case in which tumor invades the anterior part of the corpus callosum without disturbances of equilibrium, such disturbances are attributable to involvement of the frontal lobe. The astasia, or ataxia of the muscles of the trunk, which is not to be confused with the disturbance of equilibrium characterized by a tendency to fall to one side, is in Halpern's opinion related to the grasping reflex, since in both phenomena tone is increased in the flexors and decreased in the extensors.

DANIELS, Denver.

A CASE OF UNUSUAL EXTRAPYRAMIDAL MOTOR DISTURBANCE, WITH COMMENTS ON ENCEPHALOGRAPHIC FINDINGS IN CAVUM SEPTI PELLUCIDI. F. RAWAK and A. VAZ-FERREIRA, Ztschr. f. d. ges. Neurol. u. Psychiat. **152**:483 (April) 1935.

Rawak and Vaz-Ferreira report the case of a woman aged 65, who was admitted to the hospital on Aug. 15, 1934. Since 1931 she had had headache, pain in the limbs and dizziness. For about two years she noted increasing weakness of the right side of the body. Early in 1934 the headache became more intense, and she began to have attacks during which the right lower limb was very weak, with accompanying paresthesia in the right upper limb. During such attacks she felt drawn backward and to the right. On May 2, 1934, she suddenly fell to the ground. She was unable to get out of bed. She could neither walk nor stand. She showed bilateral signs of involvement of the pyramidal tract, unsteadiness of gait, with marche à petits pas, and weakness of the right lower limb. There were absence of associated movements during walking and occasionally retropulsion toward the right. There was increase of plastic tonus in all muscles. The striking feature in this clinical picture was progressive increase in the tonus of antagonist muscles during passive movements of the right upper limb. Only one other instance of a similar condition has been reported by Gerstmann and Schilder in a case of parkinsonism associated with chronic encephalitis. An encephalogram showed widening of the cavum septi pellucidi as well as bilateral symmetrical internal hydrocephalus. There was an air-filled space between the lateral ventricles and

above the third ventricle. This case illustrates the fact that the cavum septi pellucidi may communicate with the ventricles. Whether this is of pathologic significance or occurs in all cases in which this anomaly exists remains to be proved by further investigation. Rawak and Vaz-Ferreira feel hardly justified in correlating these encephalographic findings with the symptom complex. They make no suggestions regarding the etiology of this unusual clinical picture. They note the association of internal hydrocephalus with cavum septi pellucidi.

SAVITSKY, New York.

Peripheral and Cranial Nerves

PROGRESSIVE HYPERTROPHIC NEURITIS. I. J. TARASSIÉWITCH and W. MICHÉJEW, Rev. neurol. **64**:18 (July) 1935.

There are many forms of hypertrophic neuritis, some approaching the Charcot-Marie type and others the leprosy type, while variations have a number of names connected with them, such as those of Gombault and Mallet, Dejerine and Sottas, Rossolimo, Marie and Boveri, Dide and Courjon and Roussy and Cornil. Tarassiéwitch and Michéjew report two cases. In the first, that of a young man, without familial history of the disease, there were an Argyll Robertson sign, abolition of the patellar and achilles reflexes, ataxia, pains in the lumbar region and legs and weakness of the lower limbs. Within five years the nerves had grown to very large proportions; clubfoot had appeared; sensibility was affected in the distal region and there was atrophy of the small muscles of the hands, with partial reaction of degeneration. The nerves were tender on pressure. Neurectomy and rhizotomy were performed for the relief of pain. The patient died later of bronchopneumonia. Examination at necropsy showed extraordinary hypertrophy not only of the nerves ordinarily affected but of the phrenic and sympathetic nerves. The involvement extended to the roots, both anterior and posterior, and to some extent to the cranial nerves. The hypertrophy involved the connective tissue, the sheath of Schwann and the perineurium and endoneurium, but the internal structure of the nerves did not seem to be altered. The myelin sheaths showed degeneration, and the axons were involved to some extent. The optic nerve showed rather pronounced lymphoid infiltration; in fact, this was present throughout the peripheral nervous system. The spinal and sympathetic ganglia also showed marked increase in the connective tissue and thickening of the capsule of the ganglion cells. There were mild retrograde atrophy of the ventral horn cells and some atrophy of the spinal ganglion cells, with ascending degeneration due apparently chiefly to rhizotomy. The muscles showed marked variability in the size of the fibers, some being very small and some very large; collections of sarcolemma nuclei were observed here and there.

The second case was that of a girl aged 16, whose parents and seven siblings were normal. The condition had begun four years before, with weakness of the hands and then frequent headaches, irritability, gradual loss of muscular power, relatively little pain, some diminution in reflexes and normal pupillary responses. The nerves were obviously hypertrophied. The relationship of the disorder to true interstitial neuritis, on the one hand, and to diffuse Recklinghausen's disease, on the other, is not altogether established; it seems advisable to group all the transitional forms under the heading neuromegalia peripherica progressiva.

FREEMAN, Washington, D. C.

Vegetative and Endocrine Systems

ON THE CONDITIONS NECESSARY FOR THE CONTINUOUS GROWTH OF HYPOPHYSECTOMIZED ANIMALS. HERBERT M. EVANS, RICHARD I. PENCHARZ and MIRIAM E. SIMPSON, Endocrinology **19**:509 (Sept.-Oct.) 1935.

Continuous stimulation of growth in hypophysectomized animals has been accomplished by the use of more or less crude alkaline aqueous extracts of the

anterior lobe of the hypophysis. The protracted use of more purified extracts has resulted in an excellent initial stimulation of growth, but after from thirty to forty days growth stasis and an actual decline in weight have resulted. Collip has explained this failure as being due to the development of an antigrowth hormone. Evans and his co-workers, however, call attention to the fact that hypophysectomized animals, if untreated, survive for a period averaging forty days. This period of survival shows a remarkable correspondence with the time at which growth declines when such animals are treated with purified growth extracts. Attention is called to the possibility that the decline may be due to the widespread derangement of bodily function resulting from hypophysectomy. Certain animals failed to respond after from twenty to sixty days and showed an actual decline when treated with more purified extracts, but rapid and continuous growth was resumed when cruder aqueous alkaline extracts were given again. The conclusion seems to be that other hormones are necessarily administered when the cruder extracts are employed. Occasional failure with aqueous alkaline extracts was spectacularly changed by the employment of parenteral injections of dextrose. After such a procedure administration of the extract produced the customary stimulation of growth.

PALMER, Philadelphia.

HYPOGLYCEMIC HEADACHE. P. A. GRAY and H. I. BURTNESS, Endocrinology **19**:549 (Sept.-Oct.) 1935.

Gray and Burtness, stimulated by knowledge of the frequent occurrence of headache as an accompaniment of hyperinsulinism, investigated the relationship of the blood sugar level to migraine and other types of headache. Study of thirty-eight patients who complained of headache as a major symptom, twenty-two with typical migraine, revealed that the headache was usually present when the stomach was empty, that the blood level was customarily between 60 and 90 mg. per hundred cubic centimeters and that relief was obtained by oral ingestion of carbohydrate substances. The characteristic headache could be reproduced by induction of hypoglycemia. Dextrose tolerance curves of these patients showed the typical flat curve indicating a high tolerance to sugar. The authors propose an "insulin tolerance test" as a simple, safe and rapid means of measuring reactivity to insulin and as a diagnostic test. The test is performed as follows: After a fast of from twelve to sixteen hours, specimens of blood for quantitative determinations of the dextrose content are taken at intervals of thirty, sixty and one hundred and twenty minutes after the intravenous injection of 0.01 unit of insulin per kilogram of body weight. The average insulin tolerance curve of diabetic patients shows a greater fall below the fasting level than the curve of normal control subjects. The curve of hypoglycemic patients is significantly lower than that of the normal control subjects.

PALMER, Philadelphia.

ADRENAL CORTEX EXTRACT. EDWARD C. KENDALL, J. A. M. A. **105**:1486 (Nov. 9) 1935.

Kendall points out that since the classic work of Addison on adrenal deficiency the adrenal gland has occupied a position of great importance and fascination. A study of its chemical nature and physiologic function has followed the usual course of studies on the other ductless glands. The first step was the proof that the gland is essential for life. The second step was the proof that within the gland some substance or substances are present which can be separated and used in substitution therapy. The work of Hartman and of Swingle and Pfiffner conclusively established the fact that the life of adrenalectomized rats, cats and dogs can be maintained with the administration of an extract of the gland. About eighteen months before Kendall wrote his paper the separation of a crystalline fraction was reported which possessed the essential physiologic activity in dogs and in human beings. This was given with a diet that contained sodium chloride in amounts slightly higher than normal. Three patients have been maintained

with the crystalline material for periods of from fourteen to thirty-six days. When the crystals were withdrawn and the administration of the same amount of salt was continued, definite symptoms of adrenal deficiency developed. These results and those in similar experiments on adrenalectomized dogs, with the addition of the administration of sodium chloride, showed that the typical symptoms which followed the removal of the adrenal glands could be controlled by the crystalline fraction separated from protein, epinephrine, phospholipids and all other substances of either an acid or a basic nature. The author discusses the function of an extract of the adrenal cortex, its standardization and physiologic activity, the relation between the extract and thyroxine and the clinical results.

EDITOR'S ABSTRACT.

THE FORMATION OF THE CORPUS LUTEUM IS DEPENDENT ON THE ANTERIOR PITUITARY LOBE, AND NOT ON THE MATURING OVUM: THE FERTILIZED OVUM AND HORMONES. BERNHARD ZONDEK, J. Physiol. **81**:472 (July 31) 1934.

Zondek rejects the hypothesis of Claudberg and Schoeller that the maturing ovum stimulates the formation of the corpus luteum. Experiments on the ovary of the hibernating bat show that corpora lutea may be produced by means of gonadotropic extract of the anterior lobe of the pituitary gland, although the ovum is pressed to one side or may be destroyed. From this it is apparent that the formation of the corpus luteum cannot be dependent on the ovum. Direct proof is given from the follicles of the sexually mature rabbit from which he had withdrawn the ova by aspiration. After removal of one ovary and resection of ovarian tissue, so that only one follicle remained, he withdrew the ovum by aspiration. After the injection of the gonadotropic extract of the anterior lobe of the pituitary gland such a follicle is converted into corpus luteum. He concludes that for the production of corpus luteum the ovum need not be present. The formation of corpus luteum can therefore not be dependent on the ovum. The gonadotropic hormone of the anterior lobe of the pituitary gland effects ripening of the follicle and the ovum, follicular rupture and development of corpus luteum. The ovum plays no part in these hormonic events. On the other hand, since the formation of the corpus luteum is dependent only on the gonadotropic hormone of the anterior lobe of the pituitary gland, it must be accepted that the fertilized ovum exerts an impulse on this lobe, which by means of the alternation of its production of gonadotropic hormone controls the continued existence of the corpus luteum. The great changes in the production of estrogenic hormone and gonadotropic hormone of the anterior lobe of the pituitary gland following the fertilization of the ovum are neither conditional to nor consequent on every fertilization, since they occur not as a general rule but only in certain animals. The mass excretion of estrogenic hormone which has hitherto been determined only in association with a fertilized ovum can be found also in male animals of the equine species. The testes of the horse contain much more estrogenic hormone than does the placenta.

PALMER, Philadelphia.

THE POSTERIOR LOBE OF THE PITUITARY GLAND: ITS RELATIONSHIP TO THE STOMACH AND TO THE BLOOD PICTURE. E. C. DODDS, G. M. HILLS, R. L. NOBLE and P. C. WILLIAMS, Lancet **1**:1099 (May 11) 1935.

It has recently been noted by Dodds and his co-workers that an extract of the posterior lobe of the pituitary gland, when injected subcutaneously into animals or administered orally in rabbits, will produce an acute hemorrhagic lesion of the acid-bearing area of the stomach. The gastric lesion is accurately confined to that portion containing oxyntic cells; no other portion of the intestinal tract is involved. By repeated injections of either a commercial pituitary extract or an extract processed with acetone and tri-nitrophenol, chronic gastric ulcers were produced. On the fifth day severe anemia developed in the rabbits into which the extract was injected, the blood count often being reduced from 6,000,000 to as low as 1,000,000 red cells. The hemoglobin content also is reduced, but not

proportionately. The substance or substances responsible for the reaction are contained only in the posterior lobe of the pituitary gland. Exhaustive controls have been made with other tissues of the body.

The authors suggest that these observations may possibly provide an explanation for a number of hitherto puzzling phenomena. For example, in Simmonds' disease it is known that there may be atrophy of the organs of the reticulo-endothelial system. Again, in certain cases of basophil adenoma of the anterior lobe of the pituitary gland, ulceration of the stomach and erythremia have been recorded. The occurrence of acute gastric lesions after operations on the base of the brain has been recorded on several occasions.

From these investigations and clinical manifestations, it seems justifiable to consider the possibility of a hormonal connection between the posterior lobe of the pituitary gland, the stomach and the blood picture. BECK, Buffalo.

A CASE OF GYNECOMASTIA. C. I. PARHON, ST.-M. MILCOU and M. SCHACHTER, Rev. franc. d'endocrinol. **13**:351 (Oct.) 1935.

Parhon and his co-workers report a case of gynecomastia in a man aged 21. The condition began at the age of 14, a year after an attack of scarlet fever. At the time of the communication the patient was 178 cm. tall and weighed 65 Kg. Both breasts were enlarged, and the nipples were slightly umbilicated and surrounded by pigmented areolas about 4 cm. in diameter. The external genital organs were well developed. The pigmentation of the sexual organs and the distribution of pubic hair were of masculine type; there was a good crop of hair on the head, but it was patchy on the face. Both the upper and the lower extremities were well covered with hair, the chest was hairless, and in the sacral region there was a triangular zone of hypertrichosis in contrast to the lack of hair on the rest of the back. The sella turcica was of normal size and appearance. The patient is said to have had a normal sexual development, with heterosexual experiences since the age of 18½. Both breasts were amputated. Anatomic study revealed mammary glands in the form of flattened disks, 8 cm. in diameter and 2 cm. thick, weighing 170 Gm. They consisted of grayish white fibrillary tissue with islets of fat and small dark zones of glandular tissue and vessels. Histologic examination revealed an abundance of collagenous tissue, a paucity of fat tissue and signs of activity of the glandular epithelium. The picture suggested mammary glands of an adolescent female in a relatively quiescent state. The authors review the literature and repeat the opinion of Levy, who considers gynecomastia as a manifestation of a heterosexual potentiality in males. This point of view is based on the occurrence of gynecomastia combined with pseudohermaphroditism or real hermaphroditism and genital malformations, as observed by Urechia. They also call attention to the opinion of Maranon, who thinks that gynecomastia is a sign of "intersexuality" and that the mammary gland has a congenital predisposition to hypertrophy, which may occur under various hormonal or mechanical influences. All authors suggest surgical cosmetic repair. NOTKIN, Poughkeepsie, N. Y.

ENDOCRINE DISTURBANCES. ERNST HEINZE, Fortschr. d. Neurol. & Psychiat. **7**:224 (June) 1935.

Heinze reviews the recent literature (1933-1935) on the rôle played by the endocrine glands in various mental diseases from three aspects: (1) the determination of glandular functions in cases of schizophrenia, manic-depressive psychoses, epilepsy, psychoses due to dysfunction of the endocrine system and states of castration; (2) the effect of therapy with glandular substances by substitution or stimulation and in some cases by mechanical intervention, and (3) the consideration of possible or probable causal relationships between endocrinopathy and certain forms of mental disease. In general, there appears to be a relatively greater number of cases of dysfunction of the hypophyses, ovaries and testes among the

psychically disturbed than in the mentally well. More specifically, according to the analysis of 4,000 cases by Rowe, there seems to exist a positive correlation between schizophrenia and hypophyseal dysfunction and between manic-depressive psychoses and dysfunction of the thyroid and ovaries. Endocrinopathy, however, is not the exclusive factor in the production of psychoses and psychoneuroses. In Rowe's series of 2,400 cases of known endocrinopathy, only 190 patients were mentally abnormal, while of the 1,600 with normal glandular functions only 60 were psychically disturbed.

Determinations of the excretion of the female sex hormones were undertaken by Saethre. An increased output of a follicle-stimulating hormone was found in 92 per cent of cases of climacteric and postclimacteric neuroses and psychosis. In only 3 instances was an increase in the excretion of this hormone associated with an increase in the excretion of the estrogenic hormone. In all except a small number of cases of neurosis associated with oligomenorrhea or amenorrhea the excretion of the follicle-stimulating hormone and the estrogenic substance was normal. The works of Allen and Strachan suggest that there is no definite relationship between the occurrence of mental diseases and the menstrual anomalies. Various endocrine products used in therapy in such cases have given inconclusive results.

In the study of a group of 328 cases of schizophrenia, Vanelli reported states of varying intensity of hypothyroidism in 96.07 per cent. The beneficial results observed in these cases by treatment with subcutaneous injections of extracts from the thyroid, pituitary, adrenal and testicular glands lead Heinze to conclude that a general glandular hypofunction is present in these cases. Lingjaerde confirmed in part these findings by noting that the basal metabolic rate was below normal in 50 per cent of 118 cases of schizophrenia. Response to desiccated thyroid by mouth showed in most cases a slight increase in the basal metabolic rate after many months of administration. Georgi and Fels studied the excretion of the follicular hormone in normal women and in 3 with schizophrenia. In a schizophrenic patient, aged 19, with amenorrhea, no hormone was found; in 2 patients without menstrual abnormalities there was hypofunction of the ovarian gland. In Saethre's series of 22 schizophrenic patients, aged from 25 to 35, 3 with amenorrhea of four months' duration showed a positive reaction for the follicle-stimulating hormone and a negative reaction for estrogenic substance. On the basis of histologic changes in the endocrine glands in 8 cases of schizophrenia, Miyata concluded that schizophrenia cannot be considered as an expression of endocrinopathy.

In a consideration of schizophrenia, Choroschko presented 72 cases in which testicular extracts were administered. In this series clinical improvement was reported in one half of the males, and one third of the females. Galant and Zaizev independently attempted treatment for schizophrenia by subcutaneous injections of placental blood on the basis that this blood contains an excess of incretory products. The results of this therapy by stimulation are said to have been inconclusive, although the response was better in the acute than in the chronic cases. Zaizev believes that these injections were particularly effective in the catatonic states.

In manic-depressive psychoses Roggenbau observed frequent dysfunction of the thyroid gland, either hypofunction or hyperfunction. Determinations of the follicle-stimulating hormone, to evaluate the hypophyseal activity, were inconsistent. Menstrual abnormalities were frequent. The calcium content was normal in all cases.

Urechia and Abély each reported on the histopathologic picture in 1 case of manic-depressive psychosis. The accumulation of colloid in the pars intermedia of the hypophysis (Urechia) and the abnormal enlargement of this structure (Abély) led the authors to conclude that the hypophysis plays an important rôle in this disease. Dussik reported 1 case of manic-depressive psychosis in which marked clinical symptoms of hypophyseal hypofunction were observed. The father of the patient presented similar symptoms. Dussik contended that two constitutional defects may be inherited at the same time and that in this case no etiologic rôle can be ascribed to the pituitary disorder.

In the treatment for manic-depressive psychoses Georgi injected products of the adrenal cortex on the basis that in the depressed phases cholesterol metabolism is disturbed. This substitution therapy in 32 cases is said to have had good results. Intramuscular injections of lacrimal secretions were without effect. Léopold-Levi used pluriglandular preparations in 3 cases of melancholia, 50 cases of asthenic condition, 2 cases of light mental derangement and in 1 case of paranoia. The results were inconclusive. Rizzatti's recourse to thyroidectomy in 20 cases of the manic phase of manic-depressive psychoses resulted in 4 marked improvements. It is difficult to evaluate these results in the light of remissions which may occur spontaneously.

In the study of epilepsy Lenart contended that the dysfunction of the parathyroid gland plays an etiologic rôle in the production of the seizures. Parathormone eosinophilia in cases of "genuine" epilepsy showed values which deviated plus or minus 20 per cent from the normal. This author believed that these findings may be of significance in the differential diagnosis between genuine epilepsy, hysteria and tetany. Bakacz reported 1 case of epilepsy in which the injections of follicular hormone prevented the appearance of epileptic seizures which, before therapy was instituted, occurred regularly every twenty-eight days at the time of the menstrual period. From this observation the author feels justified in stating that the luteal hormone is among the mobilizing factors of the convulsions. Lederer has treated 18 patients with epilepsy with such products as an insulin-free pancreatic extract, a skeletal muscle extract and Zuelzer's liver extract on the theoretical assumption that these substances would affect the cerebral vascular spasm which is considered as the underlying cause of the convulsions. Of these patients 15 have been reported as improved. Kausch treated 2 patients with epilepsy and oligomenorrhea with ovarian hormone. The results are reported as good. Stein pronounced endocrine therapy ineffective in cases of epilepsy.

Heinze also reviews briefly a series of reports on cases in which psychotic states were found superimposed on such conditions as Simmond's disease, diabetes insipidus with acromegaly, exophthalmic goiter, myxedema and dystrophia adiposogenitalis. The effects of endocrine therapy in these cases varied; in a few instances there is said to have been improvement in the mental condition which seemed to parallel improvement in the other clinical manifestations.

Articles by Lange, Schultze and Gall consider some of the aspects of castration in relation to the causation of mental diseases. On a very limited number of cases the general conclusion prevails that castration neither causes nor aggravates mental diseases, nor is it of beneficial influence from a therapeutic point of view.

HELEN RICHTER, Boston.

Treatment, Neurosurgery

THE RÔLE OF IODINE IN THE THERAPY OF SYPHILIS. E. T. BURKE, Arch. Dermat. & Syph. **32**:404 (Sept.) 1935.

In the normal person excessive deposits of fibrous tissue are prevented by the existence in the blood of autolytic ferments which inhibit fibrosis. When these ferments are neutralized, excessive fibrous tissue formation occurs. The development of unsaturated lipoid radicals is characteristic of certain diseases, notably syphilis; these lipoids neutralize the autolytic ferment, thus permitting excessive fibrous tissue to form. Iodine becomes linked with the lipoids, making it impossible for them to neutralize the autolytic ferment, in this way indirectly, but effectively, preventing fibrosis. In every stage, this process of fibrosis must be discouraged, and there is therefore no stage of syphilis in which iodine is contraindicated. However, this drug has no spirocheticidal action and must be used concurrently with bismuth or arsenic. In fact, the fibrolytic effect of iodine is so powerful that its exclusive use might liberate previously walled-up colonies of spirochetes. Because

inorganic iodine salts are so rapidly eliminated, they should be given in large doses. Burke recommends an optimal daily dose of potassium iodide of not less than 90 grains (5.8 Gm.). Even more effective is the administration of iodine in colloidal form jointly with arsphenamine or bismuth.

DAVIDSON, Newark, N. J.

"PROSTIGMIN" IN THE TREATMENT OF MYASTHENIA GRAVIS. E. A. BLAKE PRITCHARD, Lancet **1**:432 (Feb. 23) 1935.

In 1934 Walker reported unmistakable improvement in a case of myasthenia gravis after the administration of physostigmine. Pritchard treated seven patients with myasthenia gravis with injections of an analog of physostigmine (the di-methylcarbamic ester of 3-oxyphenyl-tri-methylammonium methylsulfate). He gave atropine to counteract the effect of the drug in slowing the heart beat and peristalsis. In all cases voluntary flexion of the fingers was recorded with a dynamometer before and after the injections. In three myographic records the tension developed in the muscles on stimulation of the motor nerves were obtained before and after the injections. In each of the patients Pritchard noted definite improvement within five minutes after the injection, and within thirty minutes the degree of improvement was greater than that obtained with any other known treatment. The effect usually disappeared completely within eight hours, but a further injection again resulted in temporary recovery. In three cases in which myographic registration was recorded, clinical remission of the symptoms coincided with the change in the myogram from the form characteristic of myasthenia gravis to the normal form. Pritchard attributes the effect of the injections to the property of the drug in delaying the destruction of acetylcholine at the motor ending by the choline esterase normally present in the blood. To quote: "The results of Dale and Feldberg so strongly suggest that the liberation of acetylcholine is concerned with the normal transmission of excitation from the somatic motor nerve-endings to the voluntary muscle-fibres that an explanation of myasthenic weakness in terms of a disturbance of acetylcholine liberation or destruction seemed likely, and is now almost unavoidable in view of the remarkable effect of prostigmin both on the clinical weakness and on the characteristic myogram of myasthenia gravis."

WATTS, Washington, D. C.

Special Senses

CHEMORECEPTORS OF BLOWFLIES. N. E. MCINDOO, J. Morphol. **56**:445 (Dec.) 1934.

This investigation deals with the morphology of all so-called gustatory and olfactory organs of blow-flies (forty-seven species of Diptera, and in particular Calliphora erythrocephala) in answer to Minnich (1932), who stated that there was no morphologic information on the chemical sense organs of the tarsi of certain insects. Experiments on flies deprived of antennae and palpi demonstrated that the olfactory sense was not disturbed. McIndoo claims to have shown that the tarsi bear no sense organs except nine olfactory pores which may be stimulated by liquids at a distance of 3 mm., as manifested by a proboscis response. The reaction appeared to be conditioned by two types of stimulation, mechanical and olfactory. There was no evidence that the pores of the tarsi are gustatory sense organs.

WYMAN, Boston.

VASCULAR FORMS OF OCULAR PARALYSIS IN THE COURSE OF OTITIS AND MASTOIDITIS. J. REBATTU and A. COLRAT, Rev. d'oto-neuro-opht. **13**:565 (Sept.-Oct.) 1935.

Infection from the tympanum often reaches the point of the petrous process, and it is through this region that paralysis is produced. The third cranial

oculomotor and the fourth patheticus cranial nerve traverse a triangular space between the attachments of the tentorium, perforate the dura and enter the external wall of the cavernous sinus; hence, they are not in close relation with the point of the petrous process. The structure of the petrous bone is variable, but, in general, the compact block formed by the labyrinth is surrounded by air cavities, which communicate with the cavities of the middle ear and extend to the point. The point contains relatively large cells. In almost half the bones the structure of the point is not of the same type as that of the mastoid, but it is the same on the two sides. The abducens nerve in the posterior fossa is in relation with the middle cerebellar and internal carotid arteries and penetrates the dura just before reaching the petrous bone; the dural opening is from 3 to 4 mm. below the edge of the bone. The two dural openings are from 2 to 2.5 cm. apart. The nerve is then in immediate relation with the petrous apex and also with the inferior petrosal sinus. It then enters Dorello's canal, in which the nerve may be easily compressed by any increase in volume of the inferior petrosal or the cavernous sinus. After leaving Dorello's canal, the nerve is in relation with the carotid artery and then penetrates the cavernous sinus. The trigeminus nerve perforates the dura at the superior edge of the petrous process, at which point it is in relation with the superior petrosal sinus. The gasserian ganglion lies on the anterosuperior face of the petrous process; directly beneath Meckel's fossa the bone, when pneumatized, contains more or less well developed cells. If the cells are not developed Meckel's fossa is in direct relation with the carotid canal. The third and fourth nerves lie to the inside of the point of the petrous process and above and medial to the sixth cranial nerve. Since they are not in direct relation with the process, their involvement in the course of otitis media and mastoiditis indicates a serious complication (extradural abscess, brain abscess or thrombophlebitis).

The syndrome of Gradenigo-Lannois is seldom encountered before the age of 5 years and is rare after 40, being most frequent between the ages of 5 and 15. It is more frequent in males. There is nothing pathognomonic about the otitis. Arrest of the discharge coincident with the onset of trigeminal neuralgia speaks in favor of petrositis. If the mastoid has already been opened, lack of correlation between the amount of discharge from the tympanum and from the mastoidectomy wound is significant. Pain in the trigeminal area occurs usually at an interval after the pain in the ear has subsided; it varies in character and may be located in the frontal, temporal, parietal, occipital, orbital or dental region. Dental pains are said to indicate a particularly grave prognosis. Usually there is no modification of sensibility. The most characteristic feature of the triad of Gradenigo is paralysis of the abducens nerve. The paralysis develops rapidly and is almost always homolateral, but may be heterolateral or bilateral. In either of the last two cases it may, in general, be assumed that the paralysis is due to hypertension or, rarely, to propagation of the infectious process from one petrous bone to the other. The temperature, the general condition of the patient and the cerebrospinal fluid are usually not modified, and facial paralysis is relatively frequent. Data obtained from roentgenography must agree with the clinical findings to be of value. The evolution is ordinarily favorable; recovery may occur without surgical intervention, but usually mastoidectomy is indicated. The syndrome of Gradenigo may be caused by phlebitis of the inferior petrosal sinus, propagation of the infection by way of the caroticotympanic canal or infection of the preformed cells in the tip of the petrosa. A benign and a severe form may be distinguished. In the former type there exist simply petrous cellulitis and meningeal congestion or, perhaps, toxico-infectious neuritis, and recovery follows drainage of the mastoid cells. In the severe form the apical lesion is more serious—osteitis with extradural abscess.

Isolated paralysis of the sixth nerve may be classified as mild, moderate and severe. Acute otitis or exacerbation of a chronic suppuration is always present; a meningeal reaction may or may not be present. Only complete clinical and neurologic study can give information as to the causative lesion. In the mild form paracentesis of the drum membrane or exenteration of the mastoid is sufficient to

cause the paralysis to disappear; probably the mechanism of the paralysis is venous congestion, which cedes with drainage. The rapid evolution and the absence of trigeminal neuralgia distinguish the paralysis from Gradenigo's syndrome. Meningeal signs, when present, are probably due to serous meningitis. In the moderately severe form the lesion is located in the petrous tip. The prognosis and evolution are the same as those of Gradenigo's syndrome. The prognosis is unfavorable in the severe form. Abscess of the cerebellum was the causal agent in sixteen of eighty cases of cerebellar abscess. The sixth nerve appears to be more sensitive to hypertension than to infectious processes, but Baldenweck collected seven cases of meningitis accompanied by paralysis of the sixth pair.

Isolated paralysis of the oculomotor (third) nerve has been observed in association with thrombophlebitis of the cavernous sinus and in suppurative meningitis. Isolated paralysis of the patheticus nerve in association with otitis media is exceptional. Lapersonne reported such a case and concluded from the associated labyrinthitis and the rapid recovery that the paralysis was of reflex nature. Diagnosis is difficult, and caution is necessary in arriving at a conclusion.

Multiple paralyses of the cranial nerves almost always terminate in death. When there is paralysis of the third and sixth nerves thrombosis of the cavernous sinus is frequently the cause, although paralysis of these nerves has been observed in cases of extradural abscess near the petrous tip. Multiple paralyses may accompany suppurative otogenous meningitis or brain abscess. In paralysis of this type arising from thrombophlebitis of the cavernous sinus it is often a complication of old otorrhea, either osteitis which determines an extradural abscess and thrombophlebitis by direct extension or, most frequently, extension of phlebitis from the lateral sinus by way of the superior or the inferior petrosal sinus or the carotid sinus.

Treatment is discussed at length. Extensive exenteration of the mastoid in cases of infection of the petrous tip is indicated; if the symptoms are not rapidly relieved the tip must be opened, preferably by the superpetrous route.

DENNIS, San Diego, Calif.

VISUAL DISTURBANCES IN ASSOCIATION WITH TRAUMATIC FRACTURE AND DISCOLORATION OF THE EXTERNAL AND INFERIOR WALLS OF THE ORBIT. E. DELORD and C. DEJEAN, Rev. d'oto-neuro-opht. **14**:99 (Feb.) 1936.

In 1935 Dejean described the syndrome of the floor of the orbit resulting from malignant tumor. The case reported in this article represents the traumatic syndrome of the floor of the orbit. As a result of an automobile accident the patient received several extensive wounds of the scalp and face, accompanied by fracture of the arch of the left orbit and of the floor and external wall of the right orbit. The visual field of the right eye was diminished in the superior and temporal quadrants, the limit following a horizontal line which passed below the point of fixation. Crossed vertical diplopia was present, but only while looking downward; this was due to paresis of the inferior rectus muscle. The right pupil was dilated. Visual acuity in the right eye varied with the position of the head: On looking straight ahead at the test card the acuity was 0.1, but as the head was raised the acuity increased until it attained 0.5. With the left eye closed, visual acuity in the right eye was 0.5 on looking straight ahead; after a moment the eye was gradually raised, and visual acuity reached 0.7. There were anesthesia of the skin of the forehead and paralysis of the frontal muscle on the left side. The disturbance of the optic nerve was caused by propagation of the traumatism to the optic canal. Study of roentgenograms revealed that the line of fracture on the right corresponded to the sphenomalar suture as far as the sphenomaxillary fissure and, on the orbital floor, to the line of the spheno-ethmoidal suture. Therefore Delord and Dejean characterize this injury as a traumatic dislocation of the frontomalar, sphenomalar and ethmomaxillary osseous sutures. The paresis of the inferior rectus muscle, the variations in the visual acuity according to the degree of elevation of the head, the irregular narrowing of the visual field and the mydriasis make a unique clinical picture.

DENNIS, San Diego, Calif.

UNUSUAL SPONTANEOUS MOVEMENTS OF THE EYES AND THEIR EFFECT ON HEAD AND BODY POSTURE IN A CASE OF ALMOST TOTAL PARALYSIS OF CONJUGATE GAZE. R. KLEIN and R. STEIN, Ztschr. f. d. ges. Neurol. u. Psychiat. **153**:242 (July) 1935.

A man aged 60 complained of difficulty with vision for about four years. A left homonymous hemianopic defect was found, with complete involvement of the lower quadrants, sparing of the maculae and hemiamblyopia of the upper quadrants. When the patient paid attention to any particular object his eyes and head tended to deviate spontaneously to the right. If he tried to fixate the object more intently this deviation of the head and eyes to the right became stronger. When the head was turned passively to the left the eyes gradually moved toward the right, the head did not return immediately to the right but often remained for a time in the median position and then suddenly deviated to the right. Spontaneous forced deviation of the eyes to the right was constant, though its intensity varied. Fine horizontal nystagmus was present, with lateral movements toward the right. Passive movements of the head to the right and left were normal, though there was some resistance to passive forward and backward movements of the head. Active movements of the head in response to commands were unimpaired in all directions, though the patient seemed to bring his head farther laterally when turning to the right than to the left. Difficulty with active movements of the head appeared only when the patient was asked to fixate an object in the left field, at which time spontaneous deviation of the head and eyes to the right became marked, and the patient had some difficulty in overcoming this tendency in attempting to look and turn to the left. With his head fixed, the eyes deviated to the right if he was asked to look to the left. When he was requested to look to the right there was only an apparent voluntary movement to the right. The automatic component of such movement was more intense than the volitional. He was unable to look upward and downward. When the head was turned passively to the right the eyes moved to the left and then slowly and spontaneously back to the right. When the head was passively moved to the left the eyes turned to the right and remained there. With passive movements of the head upward the eyes moved down, in spite of absence of voluntary conjugate with downward movement. The eyes were able to follow objects to the left when the patient was constantly prodded to do so. The eyes spontaneously returned to the right if the object was too small, was covered or moved too quickly. On attempting to follow an object toward the right a spontaneous automatic movement of the eyes to the right ensued. When the eyelids were closed and the room darkened forced deviation of the eyes to the left was observed. When light filled the room spontaneous deviation to the right was seen. Bell's phenomenon was normally present on closing the lids. Convergence was absent. There were a mild left Babinski sign, masked facies, occasional forced crying and some difficulty in turning the body to the left.

Analysis of the available data reveals that with voluntary attention and fixation there was forced deviation of the eyes to the right. Klein and Stein suggest that there was hyperreflexia of the mechanism for conjugate gaze. The intactness of conjugate gaze with passive movements of the head pointed to a lesion above the nuclei for conjugate gaze in the brain stem. The left hemianopic defect suggested implication of pathways for conjugate gaze which pass from the occipital region to the brain stem. Fixation of an object is a function of the occipital cortex. There was involvement in this case of fixation pathways on the right side of the brain. The result of this lesion was that whenever the patient attempted to fixate, the influence of the pathways on the left became manifest. Bárány tests showed deviation of the eyes in the same direction as the turning, followed by nystagmus with a rapid and a slow component. The fact that the rapid component was present is evidence for a suprasegmental, perhaps subcortical, lesion and points to intactness of reflex pathways in the brain stem.

The clinical findings in this case show that the rapid component of nystagmus can be present though the eyes deviate during vestibular stimulation. They also show that the rapid component and intactness of voluntary conjugate deviation do not run parallel to and are not part of the same mechanism. The deviation of the eyes and the head and body in the same direction is the expression of a postural reflex, a tonic neck reflex beginning in the eyes. Persistent deviation of the eyes induces turning of the head and body. By injury to one component of this complex reflex mechanism latent patterns of response are released. Autopsy was not performed.

SAVITSKY, New York.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Jan. 16, 1936

C. A. McDonald, M.D., *Presiding*

Defects of Vision Following Removal of the Visual Cortex in Mammals.

Karl U. Smith, PhD., Providence, R. I.

This paper presents certain new findings bearing on the effects of removal of the visual cortex on discrimination of brightness in the cat. The validity of using this animal in the investigation of the functions of the cerebral cortex is demonstrated by preliminary statements regarding the visual acuity and discrimination of form, pattern, movement, brightness and flicker in the cat as compared with man and other mammals.

In a first series of experiments measurements of the threshold of discrimination of two lights were made at two levels of differential intensity under conditions of general low illumination (intensity of the surround), the standard intensities at the two levels approximating, respectively, 0.5 and 50 apparent foot-candles. At the low and the high levels of differential intensity, animals with both partial and complete removal of the striate cortex gave small but insignificant indications of threshold loss as compared with similar measurements made prior to operation, but no differences in the degree of threshold loss were ascertained at the two levels of intensity.

In a second series of experiments the two differential stimuli were kept at constant values (approximately 0.5 and 50 apparent foot-candles, respectively), and the effects of complete destruction of the visual areas were studied under three conditions of general illumination (intensity surround of the differential stimuli): a low (minimum), a median and a high level of general illumination. Three animals, trained in the habit of discrimination under all three conditions of general illumination prior to operation, relearned after operation the discrimination of the stimuli under the conditions of low general illumination. Approximately the same number of trials were required to establish the habit in the preoperative and in the postoperative training period. Two of the animals failed to relearn the habit when a median level of general illumination was employed, although this habit was learned within from 50 to 100 trials prior to operation. None of the 3 animals relearned the habit after operation under the conditions of high general illumination, even though from five to ten times as many trials were given as were required to learn this discrimination in the preoperative training. As compared with this, control animals, with removal of the striate cortex on one side only, quickly relearned the discrimination of the two lights under all three conditions of general illumination.

The theory is proposed that the visual cortex functions importantly in mediating responses to differences in intensity in vision under conditions of high illumination of the retina as a whole, whereas such mediation may not be demanded under dark-adapted conditions. The results also suggest that complete occipital lesions in mammals below man, instead of inducing specific defects in pattern and object vision, as has been believed by previous investigators, bring about a generalized disturbance in all visual functions under conditions of high general illumination. Further evidence in support of this point of view is cited in connection with the results of preliminary experiments on pattern vision in the cat which are being carried out at the psychological laboratory of the Brown University.

DISCUSSION

LEONARD CARMICHAEL, PH.D., Providence, R. I.: Sir Joseph Barcroft, Fellow of King's College, Cambridge, England, in a recent and stimulating book (Features in the Architecture of Physiological Function, New York, The Macmillan Company, 1934), presented the following tabulation, the factual basis for which he then adversely criticized:

	Protopathic Sense	*Epicritic Sense*
Vision	Rods	Cones
Tactile sense	Naked nerve-ending	Encapsulated endings
Tracts in cord	Spinothalamic tract	Posterior column Spinothalamic tract
Ultimate destination	Thalamus	Cerebrum

So far as I know, in this tabulation is given the only explicit suggestion of a correlation between the anatomic and physiologic duplicity of the visual receptor mechanism, established since the time of von Kries, and the protopathic-epicritic theory of Head and Rivers, although in 1929 Henschen, in an obscure Swedish publication (*Hygeia* **91**:417 [June 15]), suggested that the rods and cones, respectively, are associated with specific types of cells in the calcarine cortex.

I believe that Dr. Smith's paper may be taken as an illustration of the advance that has occurred in the technic of correlating structure and function since the time of the formulation of the protopathic-epicritic theory by Head and Rivers. Their theory was formulated to account for observations made in a single study of cutaneous nerve regeneration, but it was enlarged to cover a much wider series of facts. Since its enunciation, however, it has been subject to a series of adverse criticisms. Trotter and Davies; Boring; Schafer; Cobb, and Lanier, Carney and Wilson have, in turn, shown that the theory cannot account for the findings of more carefully controlled studies on the recovery of sensory capacity paralleling cutaneous nerve regeneration.

The facts established by Minkowski, Lashley, Marquis and many others, and especially the findings presented in Dr. Smith's paper this evening, might at first inspection lead to acceptance of the hypothesis presented in Barcroft's table, namely, that the mechanism of rods and cones is a protopathic-epicritic one. But the history of the successive revisions of the Head and Rivers theory in the hands of later experimentalists shows how wise Dr. Smith has been in not postulating so simple and, in terms of the neurology and psychology of the present decade, so naive a view of his results. He seems to have sound grounds also for questioning the fundamental duplicity of function implied in the distinction between object vision and the capacity for the discrimination of brightness.

Dr. Smith is also to be complimented, I believe, for not attempting to explain his quantitative and well controlled findings by recourse to a faculty of attention or some other purely verbal entity.

DR. T. J. PUTNAM: It is of interest to recall the development of the importance of the cortical visual apparatus. Birds manage well with only the tectal mechanism and no cortical representation. It has been known that rabbits, for example, recover useful vision after removal of the cortex, but little work has been done previously on higher animals. One cannot help wondering how far these studies can be applied to human beings. Patients may retain some visual acuity after extensive resection of the occipital cortex, but as far as I am aware a case has never been reported in which destruction of an entire striate area, verified by autopsy, was followed by return of vision, even at the macula, in the corresponding half-field. The persistence of central vision after resection of the occipital lobe, which seemed at the time of operation to include the entire striate area, has been reported by Penfield and others but this is difficult to judge. In at least one such case subsequent autopsy revealed a remnant of visual cortex.

DR. K. U. SMITH: In a review of the literature in regard to the functions of the visual cortex in man, one soon reaches the conclusion that this material is of little or no aid in solving a problem of the type approached in the present study

on cats. Furthermore, I would limit the remarks made here to the functions of the visual cortex in the cat and would not attempt to apply them to man or to other primates.

ACUTE INFECTIOUS POLYNEURITIS. DR. IRA C. NICHOLS, Providence, R. I.

The case is described of a man, aged 42, in whom twelve days after pneumonia there developed flaccid paresis of the musculature of the trunk and extremities, with facial diplegia. The tendon and superficial reflexes were abolished. Sensory impairment and paresthesias were present, being most marked in the distal parts. Recovery began on the twelfth day. The face was the last to initiate and to complete recovery. The patient has returned to his job but has none of his former energy and vitality. Examination of the spinal fluid showed a high total protein content (on one occasion, a reading of 2,000 mg. per hundred cubic centimeters) and a relatively low cell count. This finding is considered to be an instance of the albuminocytologic dissociation of Guillain and Barré.

DISCUSSION

DR. C. A. MCDONALD: This is a classic case, similar to those which Dr. E. W. Taylor and I reported some time ago.

DR. STANLEY COBB: This is an extraordinarily well handled report. I was especially interested in the relation to pneumonia, for in the cases I have seen at the Massachusetts General Hospital there has been almost always an insidious onset, sometimes after influenza. Polyneuritis of this type resembles in many ways infection with a virus. I wish to ask for opinions on that.

DR. C. S. KUBIK: In cases of polyneuritis such a high protein content of the cerebrospinal fluid is usually not shown. In 2 cases I have made postmortem examinations, although one expects recovery. The pathologic condition seems to indicate that this is an infectious process affecting the peripheral and, finally, the cranial nerve roots. I observed no evidence of involvement of the optic chiasm, the spinal cord or the brain. I think the infecting agent is probably a virus. Bradford, Bashford and Wilson claimed to have reproduced the disease in monkeys. I do not believe that any one else has been able to do so, however. Monkeys were inoculated with material from our patients, and the disease failed to develop.

DR. I. C. NICHOLS: As to the infective origin of the disease, a few reports in the literature which I have read seemed to stress more the view that it is a virus disease. No one has been able to repeat the work of Bashford and his associates. The onset follows sometimes an infection of the upper respiratory tract. One patient, whose case was reported, rode in a cold railroad cab and a few days later presented a picture typical of this disease. As to the high protein content of the spinal fluid, I would have doubted the laboratory findings if the fluid had not been so "sticky" that manometer readings could not be obtained.

LOCALIZED ANALYSES OF THE FUNCTION OF THE HUMAN BRAIN BY THE ELECTRO-ENCEPHALOGRAM. HERBERT H. JASPER, D. ÉS SC., Providence, R. I.

Since the recent statement of Gibbs, Davis and Lennox (Electro-Encephalogram in Epilepsy and in Conditions of Impaired Consciousness, ARCH. NEUROL. & PSYCHIAT. **34**:1133 [Dec.] 1935) that the method of electro-encephalography is exceedingly simple and that the technic is as free from sources of error as that of electrocardiography, I believe that it is important to point out that the method is actually exceedingly complicated and that even the most experienced electrophysiologist cannot always at present distinguish between the potentials of the brain and those of extracranial origin.

First, it must be kept in mind that the electrical potentials under consideration are extremely small electrostatic potentials, from 0.000010 to 0.000200 volt in magnitude. The same sort of potentials of magnitudes up to many volts, or one million times as great, may be produced merely by drawing a comb through

the hair. With as sensitive a recording instrument as this and with the recording electrodes separated from the cortex by several layers of poorly conducting materials, it is surprising, at first, that it is at all possible to obtain records of brain activity which are not grossly distorted or masked completely by potentials of extracranial origin. The eyes, for example, act like small batteries in the head, since the retina is positively charged with respect to the cornea. The movement of the eyes may produce potential disturbances in electrodes placed over the frontal portion of the head especially, which are much greater than normal brain potentials and of the same form as some pathologic brain potentials. Slight movements of the scalp beneath the recording electrodes may also produce changes of potentials which resemble closely some of brain origin. In fact, I have been able to simulate closely both the form and the frequency of the seizure waves in epilepsy described by Kornmüller (Kornmüller, A. E.: *Fortschr. d. Neurol., Psychiat.* **7**:391 and 414, 1935) and more recently by Berger (Berger, H.: *Arch. f. Psychiat.* **103**:444, 1935), and by Gibbs, Davis and Lennox. This was done merely by imitating the rhythmic clonic movements associated with some petit mal seizures.

Another important source of error is the fallacy of the indifferent or ground electrode. It has been assumed by some workers that if the ear, for example, is used as a ground electrode any potential disturbance obtained between this lead and a small electrode placed on the vertex originates just beneath the electrode on the vertex. Actually, under these conditions of recording, the potentials observed might well arise more nearly beneath the ear if the greatest source of disturbance was in the temporal region or if the muscles in the region of the ear were contracted.

In spite of these difficulties, it is possible to obtain a faithful record of the electrical activity of grossly localized cortical areas if the proper precautions are taken as to the placement of electrodes and sufficient controls are made to rule out any possible artefacts. Some of these controls have been summarized in a recent paper in the *Journal of General Psychology*. Records in cats were taken simultaneously from electrodes placed directly on the surface of the cortex and on the scalp directly above; a negligible distortion was found in the form of the brain potentials as they passed through the overlying tissues.

After many experiments with various types and placements of electrodes, I have concluded that maximum fidelity and localization of brain potentials may be obtained with a pair of carefully constructed electrodes placed on the surface of the scalp, from about 1 to 2 cm. apart, over a region of the cortex which is as homogeneous in structure as possible. By the use of three completely independent recording systems it was possible to demonstrate completely noninterfering localized electroencephalograms from the frontal, central, parietal, occipital and temporal regions in one hemisphere, as well as complete differentiation between homologous regions in the two hemispheres. For example, the region of origin and the path of spread of seizure waves in epilepsy may be accurately traced from one area to another. Frequently, the seizure waves at the focus of the discharge may continue, in the left motor region for example, almost incessantly, with only rarely any sign of the spread of the activity to other parts of the brain. The patient may present no clinical signs of convulsive behavior, although some confusion or dulness is often noted in his school work. All regions of the brain are usually involved in a complete attack.

Normal persons present practically the same electro-encephalograms from bilaterally homologous regions. In some cases in which lesions are predominantly unilateral, as shown by the roentgen encephalogram, abnormal brain potentials may be obtained over the pathologic brain tissue. In diffuse lesions, such as those in postencephalitic conditions, brain potentials of many abnormal types are obtained from various regions, with some potentials which fall within normal limits. There is also a marked degree of disintegration of bilaterally homologous regions, as well as of different regions of one hemisphere. These preliminary results are presented merely as an illustration of the possibilities of the technic. Detailed conclusions cannot be drawn as to any one of these pathologic conditions until more cases have

been studied and more is known of the limits of normal variation, which are recognized to be great but which do not overlap those of the pathologic electro-encephalograms discussed at this time.

The potential disturbances of brain origin which may be detected on the surface of the scalp are due apparently to the synchronous beating of large groups of cortical cells, which may in some instances take the form of a wave of activity passing over a relatively homogeneous cortical surface. The record from a given region is usually complex, composed of slow and rapid components (the alpha and beta rhythms). There is evidence from the work of Kornmüller on animals and from the work my associates and I have done on man that the slow components predominate in the occipital region in the majority of persons, while the rapid components are relatively predominant in the central region. Kornmüller suggested that these differences may be associated with the predominance of cortical cells of the granular type in the occipital region, as compared with the number of cells of the agranular type in the precentral region. This suggests that the electro-encephalogram may bear some relation to cortical cell structure.

Another important factor controlling the frequency and regularity of brain rhythms is what may be called the cortical excitatory state. This may be a more or less localized effect due to the specific sensory stimulation of a given region, or it may be a generalized effect, such as might be produced by a fairly steady state of tension involving the autonomic system or a state of relaxation in sleep. With a decrease in excitatory state the brain rhythms are slowed; they become irregular and may drop out completely, being brought back to sensory stimulation in the case of sleep. With an increase in excitatory state the rhythms are increased in frequency until, above a certain level, they again become irregular and may drop out altogether. Until these functional variations are understood more thoroughly, no definite correlation can be worked out between cyto-architectonic structure and the bio-electric potentials accompanying the activity of brain cells.

DISCUSSION

DR. H. DAVIS: I wish to express my admiration for Dr. Jasper's work, for it is easy to fall into technical error in investigations of this type. The fluctuations in potential which can be led off from the scalp may often bear little or no relationship to what is going on in the brain underneath. When I first heard of Berger's work I was extremely skeptical and was inclined to believe that his records were made up of artefacts. All who have worked for any length of time with the recording of small potentials from the body know how easy it is to be deceived by a movement of the electrodes or by pick-up from a distant muscle or a hundred other sources. The work which my colleagues and I have done has confirmed that of Berger and, in most particulars, that of Dr. Jasper. We have found, as has Dr. Jasper, that one can guard against artefacts or identify them. In trying to establish the type or types of activity which are met in normal persons, we have tried also to establish basal conditions of attention. The subject must not be in an unusually excited condition nor so drowsy that his record is essentially that of sleep. When we said that the method is simple we meant that to obtain a record of the electrical activity of the brain is simple, once certain precautions are taken; in consideration of the complexity of this organ and the inherent difficulties to be expected, this is worth comment. We did not say that the interpretation of the records of the electrical activity of the brain is simple. At present this is so difficult that we have not attempted it except in a tentative way. It is satisfactory to see records which were taken with a somewhat different instrument and which, with the extreme care that Dr. Jasper has used, look so much like our own. I take it that Dr. Jasper has been able to avoid the artefacts due to movements during an epileptic seizure, as we feel confident that we have done. I think that Dr. Jasper expressed an important fact: Further study is needed of the normal brain before one can accomplish much in pathologic conditions. Our impression so far is that normal persons tend to run true to type, giving off the same type of record hour after hour and day after day, under standard conditions. There

is a great variety of patterns among normal persons. We do not know the basis of this variation or whether it may be psychologically or clinically significant. Much more work must be done, and it is fortunate that Dr. Jasper and others are attacking the problem from various angles.

DR. F. A. GIBBS: It is of interest that by wiggling his ears, eyes and scalp Dr. Jasper has learned to imitate, to his own satisfaction at least, the wave and spike formation which we described as characteristic of petit mal. I presume that on the slide showing this artefact negative is up. If the wave and spike pattern is inverted, that is, if negative is down, the pattern appears as a series of widely spaced double spikes, which to the unpracticed eye bear little relationship to the wave and spike pattern. Dr. Jasper's records are full of these widely spaced double spikes. If he turned his records over he could not fail to see the three-a-second wave and spike which we described and which he suggests is an artefact. The difference between our records and those of Dr. Jasper appears to be due to a difference in the point of view. We always show negative as up; this has for some time been conventional in recording heart and striped muscle potentials, and it seems appropriate to abide by this convention in recording the electrical activity of the brain. Dr. Jasper shows his artefacts in a position which makes them resemble our records for petit mal and displays his records of petit mal seizures in a position which obscures their resemblance to his artefacts and, incidentally, to our records of petit mal. Whether he knows it or not, Dr. Jasper's records are in full accord with our published report, and the similarity between our records and his would be obvious to any audience if he turned his slides around.

DR. H. H. JASPER: I wish to thank Dr. Davis for his remarks and to add that my frequent discussions with him have been valuable in attempts to meet the many problems involved in research on electro-encephalography. As Dr. Davis suggests, it is encouraging to find that so much corroboration can be found in the records taken in different laboratories with different recording technics. Dr. Gibbs has raised the question of the polarity of the potentials led off from the surface of the head. In regard to the rather simple problem of recognizing the form of a given electrical disturbance when it is oriented on the records either as a deflection above the base line or as inverted, I can say only that if one is sufficiently familiar with such records their orientation is of no consequence. There is a difficult and important problem which Dr. Gibbs might have asked about, in regard to the true polarity of the electrical disturbance at the seat of activity within the brain as related to potentials observed on the surface of the head. I cannot discuss this problem at this time. I will say, however, that we have observed a complete reversal in polarity under certain recording conditions as the potentials pass from the surface of the cortex to the surface of the scalp. Consequently, one cannot be certain that a positive potential led off from the scalp actually represents a potential of the same sign at the seat of activity within the cortex.

PROGRESSIVE FACIAL HEMIATROPHY: REPORT OF A CASE. DR. WILLIAM NEWTON HUGHES, Providence, R. I.

E. W., an American high school girl aged 15, received a small laceration on the right side of the forehead at 6 years of age. She had scarlet fever in 1927. About six months before the onset of the facial lesion, her tonsils were removed. There was no history of infection or trauma of the head immediately preceding the onset of the facial lesion in November 1931, when sclerodermal patches developed beneath the right eye and on the right side of the forehead. The patch on the forehead extended into the scalp, causing alopecia. Atrophy of all the tissues on the right side of the face was soon observed. A roentgenogram of the chest, an electrocardiogram, examination of the spinal fluid, studies of the blood chemistry and the Wassermann reaction of the blood gave essentially normal results. Improvement in the scleroderma occurred when solution of posterior pituitary was injected subcutaneously, but no essential improvement has resulted in the atrophic tissues.

Recent examination showed that all the tissues—skin, fat, connective tissue and muscles—of the right side of the face showed marked atrophy, except those over the zygoma and the malar bone. The sclerodermal pigmented areas showed considerable flexibility, and hair was present on the scalp where the area of alopecia had previously been. This hair was said to have grown back during the last year and a half. The hair was of two shades of brown, with more of the light brown faded streaks on the right than on the left side. The lower eyelid was pulled down somewhat in the sclerodermal scar, showing more of the conjunctiva than on the other side. There was slight injection of the conjunctiva in the right eye and possibly more secretion of tears than on the other side. The patient ordinarily noticed no difference in the sweating of the two sides of the face. No objective sensory changes were detected. If she struck the right side of the face it generally hurt more than if she struck the left side. The four upper and the three lower medial teeth on the right were definitely shorter than the corresponding teeth on the left. The right pupil was slightly larger than the left; both reacted well to light and distance and consensually. There was slight lateral nystagmus on extreme conjugate deviation. The fundi were normal. Smell was essentially normal. The auditory nerves were normal. The tongue showed atrophy on the right, with deviation to the right on protrusion. The third, fourth, sixth, seventh, ninth, tenth and eleventh cranial nerves were normal, except as previously noted. There was an irregular port-wine mark, 14 cm. long by from 2 to 4 cm. wide, on the outer posterior aspect of the lower half of the right thigh. Roentgenograms of the skull taken on Jan. 14, 1936, showed decreased density of bone on the right, especially in the frontal and temporoparietal regions. The basal metabolic rate on Jan. 15, 1936, was minus 18.

DISCUSSION

DR. J. LERMAN: My interest is purely from the point of view of scleroderma. I began to treat patients with this condition with solution of posterior pituitary—obtaining a clue from an old Italian paper—and found that it produced marked flushing and pallor, leading to marked softening of the sclerosis. It is difficult to decide what benefit has been produced in this case—whether treatment has been worth anything. Certainly, the atrophy has not been changed. If anything, it has progressed, probably because the patient has grown. The sclerotic tissues, however, have become softer. Sometimes the tissues return to normal, but usually not; atrophy replaces the scleroderma.

DR. C. S. KUBIK: The skin was atrophic and was practically devoid of hair follicles, sweat glands and sebaceous glands. I do not think the condition was scleroderma if the hair is growing back.

DR. J. B. AYER: My knowledge of this subject is drawn from the same source as that of Dr. Hughes—from Dr. Archambault's paper (Archambault, La Salle, and Fromm, N. K.: Progressive Facial Hemiatrophy, ARCH. NEUROL. & PSYCHIAT. **27**:529 [March] 1932) and those of others. It is interesting how neurologists invade other fields and workers in other fields invade the neurologist's. Not long ago Dr. Paul Yakovlev, at the Massachusetts General Hospital, pointed out the lesions of the skin in a case and demonstrated the correlation with lesions of the nervous system. One must keep up with all aspects of medicine, but the conditions of the skin should be the easiest to study, for one can see them most easily.

DR. P. YAKOVLEV, Palmer, Mass.: I wish to make a remark concerning the possibilities of treatment. Not long ago I chanced to read an article by a Norwegian physician (Bøe, Hilmar W.: *Acta psychiat. et neurol.* **9**:1, 1934), who reported a case of facial hemiatrophy. The young woman was much disfigured and had lost her upper teeth on the side of the atrophy. For purely cosmetic reasons, Bøe used what he called an expansion prosthesis, made of india rubber, to fit in the defect resulting from the loss of teeth and atrophy of the upper jaw. A few months later he was surprised to observe marked improvement

in the trophic condition of the bone and skin on the affected side, and the vasomotor reaction, previously absent on that side, then returned (emotional flushing). His explanation of the favorable effect of the prosthesis was that the mechanical irritation and stimulation of the atrophic tissues by the prosthesis helped the circulation and nutrition and thus led to improvement. I have had no personal experience in cases of facial hemiatrophy. The relation of this condition to congenital ectodermal dysplasias, such as a neoplastic malformation of the autonomic centers and nerves, is interesting but as yet obscure. Both atrophic and hyperplastic processes are observed. I have recently had occasion to see a case of facial hemihypertrophy associated with neuroma of the facial nerve.

DR. H. R. VIETS: Dr. E. W. Taylor had a case, in which he must have taken pictures twenty years ago.

Feb. 20, 1936

H. R. VIETS, M.D., *Presiding*

ENCEPHALITIS DUE TO TRICHINA. DR. H. HOUSTON MERRITT and DR. MILTON ROSENBAUM.

Recently, 2 patients with neurologic complications of trichinosis were seen at the neurological unit of the Boston City Hospital. The first patient, a girl aged 17 years, noted swelling of the eyelids and bloodshot eyes seventeen days before her admission to the hospital—forty-five days after ingestion of pork—followed by stiffness of the neck seven days before admission and mental confusion and delirium two days before. Examination revealed spastic left hemiplegia and flaccid paralysis of the left shoulder girdle. The patient recovered and was discharged in three weeks. The possibilities of acute anterior poliomyelitis and encephalomyelitis were first considered, but the correct diagnosis was made after eosinophilia appeared in the blood; this, however, was not until six days after admission to the hospital. It was confirmed by cutaneous tests and biopsy.

The second patient, a Negro girl aged 13 years, became ill nine days before admission to the hospital, with symptoms of involvement of the eyes. She was admitted with extreme muscular weakness of the extremities and trunk and absence of tendon reflexes. She recovered after four weeks. The possibilities of acute anterior poliomyelitis and polyneuritis were considered at the time of admission. The correct diagnosis was made when eosinophilia appeared five days later and was confirmed by cutaneous tests and biopsy.

Trichinosis with neurologic complications may simulate acute anterior poliomyelitis, encephalomyelitis, polyneuritis and meningitis, but cases in which muscular weakness and absence of reflexes occur are the most common. The records of 110 patients with trichinosis admitted to the Boston City Hospital in the past twenty years were reviewed; in 11, or 10 per cent, the patellar reflex was absent bilaterally. Cases in which mental symptoms, focal signs and cranial nerve palsies occurred have been described by several authors, and summaries of these reports were given. In 9 cases in which neurologic complications were present studies were made at necropsy; nonspecific changes in the brain were observed in some, while in others there were nodules composed of endothelial, hematogenous and glia cells, in the midst of which was a trichina embryo.

Although trichina embryos and lymphocytes have been observed in the spinal fluid in some instances, examination of the spinal fluid in 12 cases in our series gave normal results, and no embryos were seen.

Trichinosis with neurologic complications must be differentiated from polyneuritis, poliomyelitis, encephalitis, meningitis, dermatomyositis and periarteritis nodosa. The diagnosis is usually made by the history of ingestion of pork, eosinophilia, cutaneous tests and biopsy. It is important to keep in mind that eosinophilia may not appear until late, as in our cases, or not at all in cases

of very severe involvement or fatal termination. Although the prognosis in the ordinary form of trichinosis is good, it is serious for patients with mental symptoms or focal signs, the mortality rate being about 40 per cent.

DISCUSSION

DR. C. A. MCDONALD: From your experience in these cases do you consider that the cutaneous test furnishes a means of earlier diagnosis than eosinophilia?

DR. M. ROSENBAUM: In any case eosinophilia may not appear till late, but it usually appears before the cutaneous reaction is positive.

DR. T. J. PUTNAM: It is easy to miss the diagnosis in cases of this sort. The diagnosis which is most often made is disseminated encephalomyelitis; this is not wholly incorrect, since disseminated lesions may be caused by an infectious agent. The cerebral lesions of trichinosis differ fundamentally, however, from those of most forms of encephalomyelitis in that the organisms are too large to pass through the arterioles; as a result, they are small and intense rather than diffuse, as are lesions of the ordinary postinfectious forms of encephalomyelitis, which appear to be due to involvement of the venous side. They resemble much more the cerebral lesions associated with endocarditis.

DR. LEO ALEXANDER: On none of the patients observed in this department was autopsy performed. Descriptions of the cerebral pathologic changes in this disease are scarce.

DR. M. ROSENBAUM: Hassin and Damon expressed the belief that the pathologic change is due to two factors: (1) toxins of the organism and of the muscle that has undergone changes and (2) an inflammatory reaction set up by the presence of the trichina embryo in the brain.

DR. D. GREGG: In a case occurring in 1909 (Acute Trichiniasis Without Initial Eosinophilia, *Boston M. & S. J.* **161**:932, 1909) there was no eosinophilia. A biopsy was made, and the trichina was observed in a piece of muscle from the calf. Eosinophilia occurred for the first time four or five days after the diagnosis was made by biopsy. I take issue with Dr. Putnam that the lesions are embolic. The organisms travel through the tissues and, to my knowledge, are seldom observed inside the blood vessels.

SIGNIFICANCE OF THE PERIVENOUS CHANGES PRESENT IN ACUTE ENCEPHALITIS ASSOCIATED WITH VACCINATION AND VARIOLA. DR. KNOX FINLEY.

This paper will appear in full in a later issue of the ARCHIVES.

EVIDENCES OF VASCULAR OBSTRUCTION IN MULTIPLE SCLEROSIS AND "ENCEPHALOMYELITIS." DR. TRACY J. PUTNAM.

This paper will appear in full in a later issue of the ARCHIVES.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 16, 1936

D. M. OLKON, M.D., *Vice President, in the Chair*

MULTIPLE SCLEROSIS SYNDROME IN A CHILD: REPORT OF A CLINICAL CASE. DR. D. M. OLKON.

A Swedish girl, aged 14 years, had complained of progressive difficulty in walking since the age of 6 years. Examination revealed good development, without apparent changes in the upper extremities. Laboratory investigations of the blood, urine, spinal fluid, etc., gave normal results.

The main neurologic findings were spastic gait and weakness of the lower limbs. The patellar and achilles tendon reflexes on both sides were 3 plus, and there was ankle clonus bilaterally. The abdominal reflexes were absent. The Babinski, Oppenheim and Chaddock signs were present on both sides, and the Rossolimo phenomenon was pronounced bilaterally. There had been two apparent remissions within three years, lasting from a few weeks to several months.

There were no signs of sensory involvement. The signs enumerated suggested clinically involvement of the pyramidal system with scattered foci in the spinal cord, hence, the diagnosis of multiple sclerosis syndrome.

DISCUSSION

DR. PETER BASSOE: Why does Dr. Olkon use the terms multiple and disseminated in this instance? The case is one of sclerosis of the pyramidal system, but why must it be called disseminated, since it appears to be limited to the tracts?

DR. D. M. OLKON: The reason I called the condition a multiple sclerosis syndrome was that there were bilateral spasticity of the lower limbs, bilateral pathologic reflexes, absence of the abdominal reflexes, ankle clonus and the two apparent remissions. One could hardly explain all these phenomena on the basis of a single focus of involvement or of any pathologic condition other than that of scattered foci involving the pyramidal system of the spinal cord.

INFLUENCE OF LACK OF OXYGEN, EXCESS OF CARBON DIOXIDE AND HYPERPNEA ON CORTICAL AND SUBCORTICAL EXCITABILITY IN MAN. DR. ERNST GELLHORN and DR. I. A. SPIESMAN (by invitation).

These investigations form part of a comprehensive program for the study of the effects of the factors of lack of oxygen, excess of carbon dioxide and hyperpnea on the central nervous system; these factors are ordinarily fairly constant in the blood but may show considerable variations under certain physiologic and pathologic conditions. In order to study the effects on the cortex in man, hearing and visual functions were studied by means of quantitative methods. The results of several hundred experiments may be briefly described as follows:

1. Experiments on hearing were carried out with the audiometer. Under the influence of from 8 to 10 per cent of oxygen, hearing was gradually diminished; the effects were reversible on readmission of air but sometimes persisted for a considerable time afterward. This course of events was not materially altered when a 50 per cent concentration of oxygen instead of air was inhaled after the period of deficiency in oxygen.

The effect of carbon dioxide (from 5 to 6 per cent) and that of a period of hyperpnea, during which the subject exhaled maximally at the rate of 35 times a minute for two minutes, were similar but quantitatively less marked than those observed in the experiments on lack of oxygen. In all three groups of experiments a supernormal phase (improved hearing) might be observed on readmission of air.

2. The effects of these factors on vision were studied by two methods: (*a*) by measuring the latent period for negative after-images obtained by a constant stimulus and (*b*) by measuring the threshold for brightness distinction with Masson disks.

Lack of oxygen, excess of carbon dioxide and hyperpnea caused a considerable increase in the latent period for negative after-images, and not infrequently even complete disappearance. The brightness distinction was greatly diminished under the influence of all three factors. The effects were reversible and were occasionally accompanied by a supernormal phase on readmission of air.

3. Psychic processes were studied by using the number cancellation test, in which the subject is requested to cross out the number 4 from a list of numbers; the time necessary for this task was determined with a stop-watch. As a second method, that of summation of two subsequent numbers in Kraepelin's tables was used. Both methods led to similar results. Number cancellations and summations were slowed by lack of oxygen, excess of carbon dioxide and hyperpnea.

4. In order to compare these results with functions involving subcortical processes, a brain stem reflex was studied in man by caloric stimulation of the vestibular apparatus by the method of Veits. In contradistinction to the results already mentioned, it was found that the sensitivity of the brain stem to lack of oxygen was slight. In some instances the number of nystagmic movements remained unaltered; in others only a slight reduction was obtained. A further distinction between the experiments on the brain stem and those on the cortex lies in the fact that excess of carbon dioxide and hyperpnea cause opposite changes in the excitability of the brain stem, whereas these two conditions produce similar results on the cortex. The number of nystagmic movements is increased after hyperpnea and decreased under the influence of excess of carbon dioxide.

On comparing the results with data available in the literature, it must be stated that the brain stem reacts to lack of oxygen, excess of carbon dioxide and hyperpnea in a manner similar to that of the spinal cord. Its reaction is fundamentally different from that of the cortex. An explanation of these findings seems to lie in the fact that, according to the observations of Cobb and his collaborators, the circulation through the brain is greatly reduced with hyperpnea. This reduction in blood flow may offset completely the increased excitability of nerve tissue generally observed with hyperpnea if, as in the cortex, the tissue is extremely sensitive to lack of oxygen. In other words, the difference between the reaction of the brain stem and that of the cortex is, in the last analysis, due to a difference in the degree of sensitivity to lack of oxygen. Furthermore, it is shown that so-called psychic processes are influenced in a fashion similar to that of the cortical processes involved in vision and hearing.

DISCUSSION

DR. PAUL C. BUCY: The findings reported are extremely interesting. However, I wish to point out that the presentation was lacking in evidence which supports the conclusion that the effects observed were due to alterations in the cerebral cortex. In fact, the authors conclude that, in regard to nystagmus as a response to irritation of the labyrinthine mechanism, these alterations in gaseous exchange produce a definite effect on the brain stem. It is difficult to see why that should be true on one occasion and not on another. It is far more likely that a deficiency of oxygen or an excess of carbon dioxide in the respired air is not without effect on all parts of the nervous mechanism—sensory end-organ, afferent nerve fibers, nuclei in the brain stem, internuncial neurons, cerebral cortex, efferent pathways and the peripheral effector mechanisms.

In connection with the experiments on hyperpnea it should be noted that an assumed reduction in cerebral circulation is not proof of the presence of cerebral anoxemia.

I wish further to ask how the authors correlate the fact that hyperpnea in suitable subjects induces evidence of cerebral excitation—generalized convulsions—with their experiments in which they conclude that the same procedure reduces cortical irritability.

DR. ARTHUR WEIL: I wish to know whether Dr. Gellhorn could recognize definite types of reaction patterns in his subjects. Did a person who was slow in regaining visual acuity following lack of oxygen also show slow recovery of hearing and response to vestibular stimuli and vice versa, or was there no uniformity in the response to different sensations?

DR. ALFRED SOLOMON: In evaluating the pathogenesis of his results, has Dr. Gellhorn taken into consideration the many changes that occur in the blood chemistry of the body, such as those in hyperventilation? On the clinical side, tetany resulting from hyperventilation is an example.

DR. LEWIS J. POLLOCK: I was a little surprised to find that soma and psyche had again been divorced and a little shocked that Professor Sherrington was thought to have denied a close relationship between the two. I wonder whether he did not mean that he could not recognize their union rather than that they had been separated.

This investigation is of great importance, but there are one or two things I wish to know. One question relates to excitability. I did not understand what was meant by visual and auditory excitability. Was this change in excitability specific for certain areas of the cortex and brain stem? These changes seemed to occur when man was subjected to lack of oxygen or to hyperventilation. The changes in an experiment with acute anoxia or loss of carbon dioxide differ from those in a chronic state of anoxia or alkalosis, and I doubt whether the same supposed changes in visual and auditory excitability would be found in chronic states. I may say that in experiments on epilepsy I have not found them. Is there not a more fundamental change, not one limited to certain sensory representations, which affects consciousness, attention, association and all other functions?

That one observes a lesser number of nystagmoid movements of the eyeballs after caloric stimulation of the labyrinth does not prove that the action of the anoxia or loss of carbon dioxide has been limited to the brain stem. I know that the diphasic reflex of nystagmus has its level in the brain stem, but a good deal of experimental proof must be adduced to show that under the condition noted there was no change in the peripheral apparatus. In the modification of diphasic reflexes, such as the scratch reflex in animals, is one dealing with diminished excitability when the response is one of shorter duration, fewer movements, etc., or is one not dealing possibly with other changes in conduction—the refractory phase, inhibition, etc.?

DR. ERNST GELLHORN: In answer to Dr. Bucy and Dr. Pollock: The increased reaction (convulsions) observed in clinical cases of epilepsy during hyperpnea does not necessarily prove an increase in cortical excitability but may be due to the fact that the brain stem and spinal cord show an increased excitability, as was indicated by our investigations on nystagmus and those of Strughold and Jöng on the patellar reflex. Dusser de Barenne obtained only slight increases in motor response after stimulation of the motor cortex during hyperpnea, and this effect might well be essentially subcortical. Moreover, electrical stimulation of the cortex is supposed to cause vasodilatation and may thereby offset the decrease in the blood flow of the brain which ordinarily accompanies hyperpnea and which seems to be the cause of the decreased excitability observed in our experiments on vision and hearing.

The interpretation of our results concerning the effect on hearing and vision as indicating a cortical action is based on the following facts: (1) the great resistance of the organ of Corti, even to complete lack of oxygen; (2) the minimal metabolism of the peripheral nerve, which allows it to continue to function normally, even in the excised condition, and (3) the extreme sensitivity of the cortex to lack of oxygen, which has been studied by numerous authors (Gildea E. F., and Cobb, S.: The Effects of Anemia on the Cerebral Cortex of the Cat, ARCH. NEUROL. & PSYCHIAT. **23**:876 [May] 1930).

It seems logical to apply the same process of reasoning to the interpretation of our experiments on vision. In the case of nystagmus the situation is still clearer, since Mowrer has shown that nystagmus occurs after the action currents in the peripheral nerve have ceased. Thus, only the central parts of the reflex arc are responsible for nystagmus (after-discharge).

An attempt to determine whether some parts of the brain stem are involved more than others has not been made, since our main point was to show the difference in the reactions of the brain stem and the cortex.

In answer to Dr. Weil's question: We have noticed that some persons are definitely more sensitive than others to lack of oxygen; i. e., changes in hearing and vision occurred with higher concentrations of oxygen than were required to produce similar effects in other subjects. Moreover, some persons showed regularly a supernormal phase after lack of oxygen, excess of carbon dioxide and hyperpnea, while others did not, which may indicate a certain pattern of response characteristic of such a person.

DR. LEWIS J. POLLOCK: I beg the indulgence of the society to clarify my question. Dr. Gellhorn did not understand my question, nor did I understand his answer.

What he said is true, if one measures the reception of the stimulus by a given method and finds that it is normal; if then, the response is diminished one can state that the effect lies in the central nervous system of the organism or the efferent system. I cannot believe that the investigators separated each of these portions of the reflex arc and examined it; so I am not convinced that there was a definite change in sensation, either vision or hearing. I do not deny that the diphasic labyrinthine reflex has its origin in the brain stem, but I do not see how Dr. Gellhorn can state that lack of oxygen acts on the brain stem just because he did not get the same number of nystagmoid movements.

DR. ERNST GELLHORN: The nystagmus was investigated by means of very weak stimuli (near the threshold). Any change in the degree of reaction seemed to be properly referred to changes in excitability, since the stimulus was kept constant.

INFLUENCE OF HYPERPNEA AND VARIATIONS IN THE OXYGEN AND CARBON DIOXIDE TENSION IN THE INSPIRED AIR ON WORD ASSOCIATION. DR. ERNST GELLHORN and DR. S. H. KRAINES (by invitation).

This paper will appear in full in a later issue of the ARCHIVES.

ACTION OF BROMIDES IN CLINICAL AND EXPERIMENTAL EPILEPSY. DR. BENJAMIN BOSHES.

A group of 72 patients suffering from epilepsy who were resistant to the ordinary bromide therapy were carefully observed, with control of the bromide content of the blood. An analysis of the response reveals that persons (43 per cent) in whom all seizures could be arrested required a lower oral dose and attained a lower reading for the bromide content of the blood than those in whom seizures persisted. Patients in whom attacks of petit mal continued required the greatest oral dose and attained a higher level of bromide in the blood than those in any other group. Patients in whom bromism developed had the highest level of bromide in the blood and were most frequent in the group of persons suffering from grand mal.

Experiments were devised on rabbits to study the action of bromides in experimental convulsions produced by intravenous injection of 2 per cent thujone in 6 per cent acacia (0.3 cc. per kilogram of body weight). It was learned that large doses of bromides (40 per cent of the total halide content of the blood) could accumulate in the blood without altering the total halide content. Bromides displaced chlorides, ion for ion, and vice versa.

Seizures produced with a standard dose of the convulsant could be ameliorated or totally arrested, depending on the level the bromide attained in the blood. The brain contained little bromide—so little that doubt was cast on the possibility that any could be recovered if it were possible to exsanguinate an animal completely and remove all nonneural elements.

The action of bromide could not be explained by the hypochloremia produced, because animals in which the amount of chlorides had been diminished were even more susceptible to induced seizures. The so-called convulsant effect of the chloride ion in patients or animals fed with bromides was shown to be due to the driving out of the bromide ion by the chloride ion, i. e., the chloride action was due to reduction of the bromide concentration.

I conclude that the effect of bromides in the treatment for epilepsy is due to the action of the bromide ion and not to a special chemical combination with nerve tissue.

DISCUSSION

DR. VICTOR E. GONDA: If I understand correctly, about 43 per cent of the patients were cured; at least it was stated that patients under bromide

management did not have seizures for years. That is such a wonderful result that I should like to hear more about it. I think one uses bromides less and gives more phenobarbital. One should cure patients if the use of bromides has such a wonderful result. I wish to know whether the patients were followed when at home. One is often told that patients have no more attacks at home, which is not always correct, for nocturnal attacks especially are frequently overlooked and likewise, the seizures which happen outside during the day and escape the attention of relatives cannot be accounted for.

DR. LEWIS J. POLLOCK: I hope I may be pardoned if I discuss the discussion. I know I should discuss the paper, but I take this liberty, for many of the patients were mine and many of the experiments were performed under my direction. I am sure that many still use sodium bromide in treatment for epilepsy and have used it for many years. I have used it largely since I began neurologic practice. I know that Dr. Hugh Patrick used it throughout his practice, until he retired. I should say that the reduction in the number of patients who had seizures to 47 per cent is a small number. Dr. Patrick always insisted on complete cessation of attacks and administration of the medicine for at least three years before its discontinuance. I am not giving a numerical figure, but I may say that I take more pleasure in the treatment for epilepsy than in that for any other condition. It does not make much difference whether one uses sodium bromide, barbital or something else if it is given intelligently and if one insists that the patient have sufficient exercise and a careful diet, watches him carefully and is sure that convulsions cease. With carefully administered therapy the convulsions cease in many instances, and if the medication is not stopped until some time later they remain absent. I do not mean that I do not use barbital at all, but I usually prefer sodium bromide. This group of patients was selected from a large group in which treatment was given.

Dr. Boshes did not use the word cured in his paper. He stated that the convulsions remained absent for varying periods. I assume that a patient would prefer to take sodium bromide regularly and remain free from convulsions than to have convulsions.

I am sure that large doses of sodium bromide continued over many years do not produce brutal effects and that no mental deterioration results from the use of the bromide. When mental deterioration occurs it is due to the effects of the disease. I am sure that administration of sodium bromide will be as successful in the hands of others, if used intelligently. How carefully it should be watched is illustrated by this incident: We wished to know its effects on patients in the clinic; so we studied a group, using 22 grains (1,425 mg.) a dose, solution of potassium arsenite and infusion of Adonis vernalis. We examined the blood some weeks after this treatment had been continued, and the bromide content was below 75 mg. per hundred cubic centimeters in most instances. The patients were not taking the medicine.

Dr. Boshes has not described a miraculous cure. He studied certain observations in a practice in which patients with epilepsy are treated with sodium bromide. He picked out a group of patients who were recalcitrant to treatment and divided them into three groups. In one group, in which attacks of grand mal could not be stopped, many patients took large doses, and bromism developed. In the group of persons with attacks of petit mal bromism was not shown; the patients required large doses, and the attacks did not stop in many instances. In the group of patients who responded to treatment, the average amount of medicination was less, and bromism was rare. Dr. Boshes showed the relation of the amount of bromide in the blood and the efficiency of treatment.

DR. BENJAMIN BOSHES: I wish to mention a statement in the literature. I have shown that most cases of bromism occurred in the group of patients with grand mal. Studies of the living cell membrane reveal that it is selective to various substances. When the brain of a bromized animal is stained by the Cajal gold method, retained bromide should fix the gold in situ. This method will show whether or not the bromide is in the brain cell. The bromide, however, is observed

around, not within the glia cells, and especially around the protoplasmic astrocytes. If it does not enter the cell one cannot assume that bromide combines with the brain substance. A given dye cannot pass through a living membrane, but if one changes the conditions by altering the p_H or by some other means, such as trauma, the dye may penetrate. It is possible that in the seizures of grand mal there may be sufficient alteration of the cell membrane that some bromide enters the brain cell and that therefore one sees a greater percentage of bromism in the group of patients with grand mal than in those with petit mal.

DR. MEYER SOLOMON: I wish to know whether the Ulrich plan of decreasing the salt intake of patients with bromide therapy was used in children.

DR. BENJAMIN BOSHES: It is well established that if an organism is fed with bromides and chlorides simultaneously it will take the chlorides chiefly. It prefers the chlorides and will take them out of any mixed solution. If the chlorides are stopped it will take the bromides. This is what Ulrich did. He used diets low in chlorides, a preparation of sodium bromide combined with bouillon in soup, etc., with control of the chloride-bromide balance, so that he obtained high bromide levels in the blood despite low oral intake. He gave sufficient chlorides in the form of medicine or food to prevent bromism. The situation in children is different. It is known that children have a low renal threshold to bromides, whereas adults have a high threshold. Children excrete bromides rapidly because they do not reabsorb the salt in the kidney as well as adults and therefore lose it in the urine. They can tolerate a much higher dose than adults. Ulrich's method is applicable to young and old, but one must keep in mind the difference in the ability of adults and that of children to tolerate and excrete bromides.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 24, 1936

FRANCIS C. GRANT, M.D., *President, in the Chair*

THE SURGICAL TREATMENT OF PORENCEPHALY: PRESIDENT'S ADDRESS. DR. FRANCIS C. GRANT.

In the past five years I have encountered a small group of 4 cases in which definite symptoms of porencephaly were presented, which, on further investigation, seemed to be based on pathologic changes so extensive that any relief with surgical treatment was considered highly improbable. The results of operative intervention in the first case, however, were so satisfactory that similar treatment was attempted in the other 3 cases with, if not complete success, at least definite improvement in the condition of the patient. I admit that in 2 of the 4 cases the time since operation is too short to determine whether improvement will be permanent.

The lesions causing epilepsy are many and varied. Generalized or idiopathic epilepsy is the result of conditions so widely diffused through the brain that available surgical methods have little or no place in their treatment. But focal or jacksonian epilepsy is another matter. A circumscribed lesion in a definite area of the brain is the cause of this condition. The brilliant work of Jackson, Gowers, Foerster and many others has established this on a firm foundation. A discrete scar in the cortex results most frequently from a localized trauma. Foerster and Penfield showed that a vascular plexus forms about such a cicatrix, with disappearance of adjacent nerve fibers. As the scar contracts, the ventricular system may be distorted, and one or both ventricles may gradually be drawn toward the scar. Gentle traction on the scar or electrical stimulation of the adjacent portion of brain may initiate a typical focal convulsive seizure. These authors

showed that surgical excision to the ventricle of such a scarred area will, in about 60 per cent of cases, result in cessation of the fits.

The 4 lesions to be described in this paper differ in their pathologic structure and are probably due to etiologic factors other than trauma. In none of these 4 cases could a history of difficult labor or subsequent cranial injury be discovered. Unfortunately, the records contained no mention of the mother's health prior to the birth of the patient. Each patient, however, had a large porencephalic cyst, which in 3 of the 4 instances was unaccompanied by other ventricular distention. The fourth patient showed definite hydrocephalus as well as porencephaly.

Porencephaly was first described and named by Heschl in 1859, although Audry, in a review in 1888, stated that the defects in the brain described by Reil in 1812 were of this nature. Kundrat, Schattenberg, von Rahlden, Schutte and Salzmann reviewed the literature and theorized on the cause of the cystic formation. Various forms of porencephaly have been described. It is generally accepted that the cyst may communicate with the ventricle or be separated from it by a thin membrane. Disease of the brain in the fetus, inflammatory processes during intra-uterine or extra-uterine life, embolism and thrombosis, primary internal hydrocephalus and primary disturbances of development, with secondary hydrocephalus and intra-uterine or extra-uterine trauma, have all been held accountable for the formation of the cyst. Disturbances of nutrition in the mother, anomalies of placental development and cramplike contractions of the uterus are assumed to produce ischemic necrosis of the brain and then porencephaly by resorption. Globus, in a recent report of an autopsy, described definite evidence of encephalitis, to which he attributed the defect. There is no evidence in any of the cases in my series to throw light on the etiology, although trauma seems to have played no part in the formation of the cyst. In a recent case in Dr. C. H. Frazier's clinic, not included in this report, hemiatrophy of the bones of the skull, recently alluded to by Dyke and Davidoff, accompanied the condition. In this case the cyst may be considered part of a congenital defect of the brain and cranial bones. It must be obvious from this brief review that, although many theories as to the causation of this condition have been advanced, little is known as to its etiology.

In recapitulation, then: Porencephaly may be congenital or acquired. The anomaly is certainly not inconsistent with life; in fact, the person with such a brain may have lived many years without any sign or symptom of the lesion. In other instances there may be marked disturbances of motor function or mentality, or the condition may cause death shortly after birth. This wide range of consequences is apparently connected with the site and extent of the defect.

(The case histories, results of the physical and neurologic examinations, observations at operation and treatment were described in detail.)

It is curious that in 3 of the 4 cases the porencephalic cyst should have been located in the left side of the brain. Review of the available literature on porencephaly did not suggest that one hemisphere is more likely to be involved than the other. The large destruction of cerebral tissue and its replacement by cerebrospinal fluid were accompanied by remarkably little motor or sensory disturbance, considering the size and position of the lesions. Complete contralateral hemiplegia rather than slight weakness and mild spasticity could well have been anticipated. The destruction of the brain and its replacement by fluid must have been slow, thus enabling adjacent centers to take over the function of the ablated areas.

Why should a porencephalic cyst cause focal epilepsy? Certainly, there is no scar tissue to tug on overlying cortical centers. The cortex covering these cysts was so thin and atrophic that it must have been functionless. If the irritation producing focal epilepsy was caused by traction on adjacent areas, the removal of the thin wall of the cyst could hardly have altered conditions sufficiently to produce this stimulation. In 2 of the 4 cases the thin layer of cortex overlying the cyst was highly vascularized. A vasomotor reflex might conceivably have affected neighboring sensitive centers and produced a fit. But in the other 2 cases the outer wall of the cyst was practically avascular. In all 4 cases the choroid plexus in the underlying ventricle was clipped, in 1 instance in both lateral

ventricles. It seems hardly probable that the slight interference with filtration of the cerebrospinal fluid could have accounted for the cessation of convulsions.

From these 4 cases certain conclusions of diagnostic value may be drawn. If a patient has unilateral atrophy and slight spasticity and focal convulsions occur in the hemiparetic limbs in the absence of any definite and cleancut history of cranial trauma, the presence of a porencephalic cyst should be suspected. At times hemiatrophy of the bones of the skull is demonstrable in a roentgenogram. As in the case of jacksonian convulsions in which there is no evidence of intracranial pressure, encephalography should be performed. Studies with the use of air will immediately confirm the presence, size and shape of the porencephalic cyst. It is my opinion that making of an osteoplastic flap, with removal of the thin membrane overlying the cyst to the adjacent apparently normal brain tissue, is indicated. The operation involves the patient in no particular risk. The results obtained in this small series of cases seem to justify surgical intervention.

DISCUSSION

DR. ROSS H. THOMPSON: The small size of the skull at the site of the cyst in 1 case may be of diagnostic importance. The skull expands under the stimulus of the growing brain. This may indicate that in this instance there was no growing brain to cause expansion. It may be that this cyst was present in prenatal life and was a fault in embryonic development. If a cyst had developed in a normal brain the mass added to the intracranial contents would have been expected to influence the expansion rather than the failure of development of the skull in that region, whether prenatal or postnatal.

DR. TEMPLE FAY: Dr. Grant mentioned that the appearance of the inside of the porencephalic cyst resembled stringy blood vessels and was similar to that of the sea-nettle. My impression in a similar case was that this network of fibers looked like the inside of a pumpkin. On examination Dr. Ernest Spiegel observed that this tissue was inflammatory and not vascular. The network Dr. Grant described, however, may have been of another type. I have been making encephalographic examinations since 1926 and now have records of more than 900 cases. Porencephalic cysts are not uncommon. I recall that I performed operation in 3 of these cases. As Dr. Grant has pointed out, almost one half of the hemisphere may be involved, and still the patient may show little disability. I am not prepared to say that the cyst has nothing to do with the convulsions per se. Convulsions indicate merely less inhibitory control on the part of the higher centers, and portions of these centers must have been involved in such an extensive process. It may easily be that convulsive seizures occur from other causes, the cyst itself only enhancing such a possibility, as convulsions arise from various types of pathologic changes in appropriate areas.

Dr. Grant recalls to my mind a case reported by Dr. J. M. McConnell, that of a woman of 59. She had left hemiparesis of the atrophic type, in which the arm did not develop. She gave a history of having had acute infection of the brain. At the age of 8 she had had difficulty in walking and going up and down stairs. At the age of 59 she fell down stairs and struck her head on the cement floor of a basement. This was followed by jacksonian convulsions in the involved arm and hand. Muscular atrophy and contractures, which produced excruciating pain whenever the seizures occurred, were present. Dr. McConnell thought that injection of the peripheral nerves of the arm or section of the brachial plexus might be necessary. I did what Dr. Grant has described in this paper—exposed the cortical area, opened a large subarachnoid cyst and closed the wound. The patient has now lived for four years without further convulsive seizures; more important, there has been some additional power in the arm and leg, and she has much less spasticity.

I wish to ask Dr. Grant what he thinks becomes of the fluid which is liberated from the ventricles and subarachnoid space and converted into subdural fluid? Has he made encephalograms after resection of the cyst? I am sure he will be as startled as I to find that the margins of the brain look like Niagara Falls, as

they float free in a mass of subdural air. In many cases of a normal brain I have found more subdural fluid than I realized. Subdural fluid, I believe, escapes by means of the extracranial lymphatics. Alexander in 1906 described a method of puncturing the arachnoid, creating direct access of fluid to the subdural space, and reported improvement in the number of seizures in about 40 per cent of cases in which operation was performed. I believe that removal of the cyst may not be the real reason for the beneficial results reported. The subdural communication established by uncapping the cyst may play a part.

DR. MAX ABRAMOVITZ: A boy aged 15 years had been born by a difficult delivery and had had convulsive seizures during the first year of life; there was no recurrence of seizures until 11 years of age. Examination showed atrophy in the right arm, incomplete right homonymous hemianopia and no changes in the fundi. An encephalogram revealed a porencephalic cyst in the left side. This case was similar to those described by Dr. Grant. The question of surgical intervention did not arise.

DR. F. H. LEAVITT: In 1920 Dr. William J. Taylor, of the Philadelphia Orthopaedic Hospital, performed craniotomy on a boy aged 8 years, who had jacksonian epileptiform attacks on the right side. After reflection of the bone flap and cutting through the dura, it was observed that there was complete absence of the left frontal lobe, extending back practically to the fissure of Rolando. In the large cyst were myriad "cobwebs," extending from the meninges to the base of the cyst. These cobwebs and the adhesions from the meninges to the cortex in the region of the fissure of Rolando were removed. During the following eight years the patient was free from attacks. No observation has been made during the past eight years. An interesting feature in the case was that despite the complete absence of the left frontal lobe the patient had practically no mental defect and was able to continue in school and keep up with his classes.

DR. S. N. ROWE: From the standpoint of diagnosis, it may be stated that the cyst does not always have a large connection with the ventricular system. In a case about a year ago, the first encephalograms showed no evidence of a cyst, but there was a great deal of air in the subarachnoid space. In films taken twenty-four hours after the first encephalogram evidence of a porencephalic cyst was discovered. In a similar case, while the cyst was visualized well in the films, the connecting opening could not be seen at operation. When encephalograms are made in cases in which a cyst is suspected it is worth while to take films twenty-four hours later.

DR. BALDWIN L. KEYES: One of the surgeons in 1921 reported to this society a case somewhat similar to these described tonight, in which jacksonian epilepsy had developed. The skull was opened, and a large cyst was observed. The surgeon resected a pad of fat from the buttock and filled the site of the cyst. The patient made a good recovery and when observed two years later had had no more convulsions. Possibly, this technic may have a point of value in preventing the falling in of the unsupported upper edges of the cystic wall, which Dr. Fay mentioned.

DR. F. C. GRANT: In regard to the question of what becomes of the fluid: A great deal about the subdural space is unknown. Penfield froze the heads of dogs, cut through the skull and observed this curious yellow fluid. As one begins to lift the brain in operating for the relief of tic, much fluid escapes without pressing long, when the dura is nicked. How did it get there? I believe that it is squeezed out of the subarachnoid space. I do not know what becomes of this fluid. I think that the same condition occurs as when a cortical or a cicatricial scar is excised. Within ninety-six hours the space becomes covered with a layer of endothelial cells. The pull on the cortex is avoided, and remission of convulsive seizures may result. In cases in which a large area of cortex is excised and the fluid is permitted to pass into the subdural space, with collapse of the adjacent brain, the cerebrospinal fluid escapes into the subdural space. One probably can connect the subdural space with the ventricle. Why that should

relieve convulsive seizures I have no idea, unless it is that cortical pull is avoided. In 2 of the cases in my series the procedure was more successful because there was considerable vascularity in the roof of the cyst. Penfield suggested that epileptic seizures may be due to vasomotor change. This vascular bed may have been subject to such changes and hence may have been the cause of convulsive seizures.

CEREBRAL ANOXEMIA OCCURRING IN FULMINATING SEPTICEMIA. DR. H. E. RIGGS.

My colleagues and I have been interested in a condition in children which is characterized by abrupt onset, with frequent convulsions, signs of respiratory and cardiac collapse and death within from twenty-four to forty-eight hours. There have been 30 of these cases in the Philadelphia General Hospital in the past five years.

The age may vary from 6 months to 15 years (the great majority of patients are under 5 years). A typical history is as follows: The patient was a sturdy, well nourished child, who had had a mild infection of the upper respiratory tract or acute diarrhea. He ate normally in the morning but vomited soon after and passed into a series of violent convulsions. Physical examination showed labored, rapid respirations, with rapid, weak pulse and peripheral cyanosis. Except for frequent convulsions the patient was completely comatose. Occasionally nystagmus was present, or there was paresis or involuntary movements of an extremity. The temperature rose rapidly to from 106 to 108 F., and death occurred, apparently from cardiac failure. Postmortem examination showed only acute congestive heart failure, with severe myocardial degeneration. Blood cultures were usually positive for hemolytic Streptococcus or Staphylococcus.

Grossly the brain was intensely engorged, with diapedesis of red cells along the subarachnoid vessels. There was intense edema, with obliteration of the convolutional markings.

Histologic study showed no evidence of inflammation but primary degeneration of the walls of the cerebral capillaries, with transudation of serum resulting in perivascular and pericellular edema. As a result of the edema there was severe degeneration of the nerve elements. Although the vascular changes were constant throughout the brain, the effect of the edema varied in different parts of the brain —apparently directly with the metabolic rate of the area. Thus, the greatest degeneration occurred in the third and fourth layers of the cortex, the cerebral vegetative centers of the diencephalon and medulla, the extrapyramidal system and the olivocerebellar system (in young children the cortex shows by far the greatest damage). There was little glial reaction except in the form of subependymal proliferation, mainly around the lateral ventricles. The choroid plexus, by the nature of its construction, was severely degenerated.

From a study of several cases of this condition, I believe that in septicemia there is a specific degenerative effect on the cerebral vascular endothelium and that, as a result of the ensuing edema, cellular asphyxia occurs. Death is due to the effect of the asphyxia on the cerebral vegetative centers.

What cerebral changes occur in patients who survive the acute period? We have studied the brains of 4 such patients who survived six weeks, five months, three years and twenty-six years, respectively. In all there was severe degeneration in the cerebral vascular system, with intense proliferation of endothelial cells. In 2 cases this amounted to progressive occlusion of some of the arteries of the subarachnoid space and cortex, with resulting fibroglial scars. In 3 patients there was mental obtundity, with marked distractibility of attention. Two of the 3 patients had convulsions at intervals during the entire period of survival. The last case is worthy of special mention.

The patient was said to have had spinal meningitis at the age of 18 months. He recovered, and development appeared to be normal. However, there was partial deafness, and the pupils were always widely dilated. Six weeks before death he was found unconscious and thereafter had a series of attacks of syncope. These

were preceded by stiffness and shaking of the hands, cold sweat and pallor of the face. On his admission to the hospital, the patient had complete heart block and died in an attack of syncope. The heart showed no changes in the auriculoventricular or sino-auricular nodes (which were studied by means of serial sections), but the appearance of the heart muscle suggested changes such as result from denervation. The cervical sympathetic ganglia showed severe degeneration of long standing and of progressive nature. There was marked, progressive degeneration of both vagus nerves, both within the skull and in the neck. The brain showed marked dilatation of the intracerebral vessels and degenerative changes in the walls, with accompanying severe cellular change. There was demyelinization of the periventricular gray matter around the third and fourth ventricles, especially in the nuclei of the tuber cinereum and the dorsal vagal nuclei. The cortex showed progressive demyelinization, with many calcified ganglion cells and many focal areas of loss of ganglion cells in the third and fourth layers. There was secondary degeneration in the descending pathways from the nuclei of the tuber cinereum and the vagal nuclei, showing that the changes in the vegetative centers preceded the terminal period of heart block.

In this case, we expressed the belief that the heart block resulted from the effect of the cerebral vascular damage to the vegetative centers of the tuber cinereum and medulla and that the cause of this vascular pathologic change was the so-called spinal meningitis in early childhood. The pericellular edema resulting from the increased capillary permeability caused local accumulation of carbon dioxide from cellular metabolism and prevented oxygen from reaching the cells. At first, this was not sufficient to stimulate the vegetative cells, but, since the condition was progressive, the accumulation of carbon dioxide in time caused a stimulating effect on the vagus nerve, thus slowing the heart. Death was caused by the superimposed effect of the resulting circulatory stasis.

DISCUSSION

DR. F. C. GRANT: What did lumbar puncture show, if any was done?

DR. H. E. RIGGS: The spinal fluid was always under increased tension and was frequently bloody, owing to the escape of red cells by diapedesis. The globulin content was slightly increased, but the cell count was never over 5 or 6 cells.

The white cell count of the blood was rarely high, suggesting that the infection was so fulminating as to suppress leukocytosis.

DR. TEMLE FAY: Did I understand that the patients who survived all had a positive blood culture? Did you observe in the group of patients who recovered portions of the brain which were shrunken and atrophic or any gross manifestations of the effect of the infection?

DR. H. E. RIGGS: Blood cultures were positive in the acute attack in 2 cases. In the other 2 instances they were not made.

Mild cortical atrophy, especially over the frontal pole, with thickening of the pia-arachnoid and lakes of spinal fluid, was observed in the more chronic conditions.

DR. ROSS THOMPSON: Do you believe that the degeneration of the sympathetic ganglion and the centers of the vagus nerve operated for several years and damaged the heart?

DR. H. E. RIGGS: I think that the "spinal meningitis" in childhood was responsible for initiating degenerative changes in either the sympathetic centers or the vagus nuclei or both, which were progressive. At first this caused no symptoms, or rather no recognizable symptoms, but finally it caused vagal stimulation, slowing of the heart and terminal exhaustion.

In any condition in which edema of the brain resulting from stasis is present over a sufficiently long period, there is progressive demyelinization of the vagal centers and the vagus nerves. This is shown in cases of long-standing congestive heart failure. In these instances sudden death without evidence of coronary occlusion or cardiac dilatation is, in all probability, due to vagal exhaustion.

DR. B. J. ALPERS: It is difficult for me to conceive of this process as due to anoxemia. It seems to me that this is giving the term anoxemia too broad a connotation; it would be a mistake to call any interference with the nutrition of the cell anoxemia. Can one not define the situation in this disturbance in other terms, for to state that the fundamental difficulty was anoxemia is to infer that one knows specifically the basis of the difficulty?

DR. H. E. RIGGS: The term cerebral anoxemia may be misleading because of the connotation with lack of available oxygen. However, in all the cases mentioned the histologic picture in the brain was identical. Owing to pericellular and perivascular edema, there was inability of the cell to discharge metabolites or receive oxygen from the blood.

In other words, the histologic picture is the same in cases of various types, but the exciting cause may be any one of a large variety of conditions, ranging from congestive heart failure, septicemia and massive collapse of the lung to botulism, diabetic acidosis and carbon monoxide poisoning. Toxic encephalitis could not account for the cerebral changes in cases of carbon monoxide poisoning, massive collapse of the lung or congestive heart failure, and yet the histologic pictures are identical.

If one calls the changes in the brain in association with streptococcic septicemia, toxic encephalitis and massive collapse of the lung cerebral anoxemia when the cerebral pathologic pictures are identical, one must admit that the neuropathologic diagnosis is not obtained from study of the brain but from the clinical studies and the history.

It is again a question of defining the terminology. If it is admitted that the brain reacts to any stimulus in a stereotyped and nonspecific manner, would it not be more logical to include in one general group cases of all types in which cerebral edema secondary to capillary damage occurs, with a qualifying phrase suggesting the origin?

LOS ANGELES SOCIETY OF NEUROLOGY AND PSYCHIATRY

Regular Meeting, Feb. 10, 1936

EUGENE ZISKIND, M.D., *President, in the Chair*

EPILEPSY AND THE CAUSES OF CONVULSIONS. DR. STANLEY COBB, Boston (by invitation).

Dr. Cobb discussed epilepsy, defining it as a cerebral syndrome in which a disturbance of consciousness overwhelms the victim suddenly and usually recurs in any one victim in a rather stereotyped pattern. It is often preceded by a sensory warning (aura) and is accompanied by motor discharge through the muscles (convulsion).

The causes of convulsions were shown in a chart, and a list was presented of 61 pathologic states in which fits may occur (Cobb, Stanley: Concerning Fits, *M. Clin. North America* **19**:1583 [March] 1936). These states were explained on one or another physiologic basis, and 11 possible physiologic mechanisms which might go beyond normal limits and become pathologic processes were discussed. These 11 processes were: direct irritation, congenital defect, tissue destruction, increased intracranial pressure, cerebral congestion, hydration, dehydration, vasoconstriction, changes in permeability, asphyxia and alkalosis. It was argued that the physiologic mechanisms which may become pathologic and disturb the brain are so varied and at times so opposite (e. g., hydration and dehydration, vasoconstriction and cerebral congestion, asphyxia and hyperventilation) that a convulsion must be a simple fundamental manifestation of nervous disintegration. The dis-

integration can be caused by so many etiologic factors that a convulsion is merely a symptom that may come from 60 or more causes, and epilepsy is in no sense a disease.

Under the heading of treatment, the various possible ways of combating abnormal physiologic processes were discussed. It was emphasized that in all cases encouragement is important and that in a certain number of instances intensive psychotherapy will be found remarkably effective. It was not thought that epilepsy is psychogenic in the ordinary sense of the word but that emotional disturbances due to environmental difficulty, which are often repressed, can be relieved. This will often lessen the nervous tension of the patient and cause a reduction in, or even a cessation of, seizures.

Treatment with neutral red, which is still experimental and must be used with great care, has been used in the cases of 2 patients at the Massachusetts General Hospital. Both were feebleminded boys, who were having from 20 to 40 seizures a day. Both have improved remarkably.

Book Reviews

Gefässmissbildungen und Gefässgeschwülste des Gehirns. By Bergstrand, Olivecrona and Tönnis. Price, paper, 24 marks; cloth, 26 marks. Pp. 178, with 137 illustrations. Leipzig: Georg Thieme, 1936.

This book is a cooperative undertaking, each author being responsible for and signing certain chapters. The work begins, after a preface by Tönnis, with a pathologic study by Bergstrand. This is concerned mainly with terminology and adds little to the present knowledge of the pathologic features of the conditions mentioned. In view of his contention that all the lesions discussed are malformations and none of them tumors, the title of the monograph is somewhat surprising. Bergstrand comes to the conclusion that the best classification is: (1) angioma cavernosum and (2) angioma racemosum, the latter being subdivided into (*a*) telangiectasis, (*b*) Sturge-Weber's disease, (*c*) angioma arteriole racemosum, (*d*) aneurysma racemosum venosum and (*e*) aneurysma arteriovenosum.

The distinguishing characteristic of the angioma cavernosum, according to Bergstrand, is the absence of cerebral tissue between the vessels. In the angioma racemosum there is cerebral tissue, usually gliosed, between the vessels. The telangiectasis is composed of capillaries, and the angioma arteriole racemosum theoretically of arteries, although Bergstrand doubts that such a condition exists. The distinction between aneurysm racemosum venosum and aneurysma arteriovenosum, according to Bergstrand, is physiologic and not pathologic. In discussing Sturge-Weber's disease he stresses the now well established fact that the calcification is in the cortex of the sulci and not in the overlying tangle of veins. His discussion of the Lindau tumors follows established lines, but he promises some embryologic studies which will clear up their origin. He stresses also that these tumors are always of leptomeningeal origin. He discusses also the problem of the angioglioma and expresses the belief that it is merely a degenerated astrocytoma with abnormally developed vessels. In chapter 3, Olivecrona gives a summary of the material and points out that the most important forms of vascular lesions from the clinical point of view are: (1) the aneurysma arteriovenosum and (2) the Lindau tumors. These vascular malformations constituted 3.9 per cent of his material, there being twenty-two aneurysms and twenty Lindau tumors. Chapter 4 is concerned with five cases of the Sturge-Weber syndrome. Chapter 5 contains an illuminating discussion by Tönnis of the aneurysma arteriovenosum. He details a number of interesting cases and points out that the only characteristic symptoms are epilepsy and the intracranial bruit. These may both be absent, however, and the diagnosis can then be established only by arteriography. He points out again that in attempting to deal with this lesion surgically one must beware of the veins. It may be possible to extirpate certain small ones, but in dealing with the larger ones the blood supply may be reduced by tying the carotid artery. This Tönnis has done in three cases; one patient died of hemorrhage a year later; in another case injection could afterward be made into the lesion from the other carotid artery; the third was done very recently. The logical method of dealing with these lesions would be to tie the feeding artery; this Tönnis has been able to do successfully in one case. Unfortunately, there are usually several feeding arteries. Tönnis is much less optimistic about the treatment of the venous aneurysm, which he discusses in chapter 6. Olivecrona discusses the Lindau tumors in chapter 7, pointing out that they constitute 13.4 per cent of cerebellar tumors. The average age of his patients was 37.5 years. Two of the patients died before operation; two died after ventriculography and two after operation. In thirteen cases the tumor was successfully removed and the patients were cured.

Undoubtedly the high point in the monograph is chapter 5. The use of arteriography has enabled the authors to make a real contribution to the knowledge of these rare but interesting lesions.

Practical Aspects of Psychoanalysis: A Handbook for Prospective Patients and Their Advisors. By Lawrence S. Kubie, M.D. Cloth. Price, $2. Pp. 223. New York: W. W. Norton & Company, 1936.

No other treatise on psychoanalysis which has come to the reviewer's attention concerns itself exclusively with the subjects elaborated in this book. They are the considerations which are specific for psychoanalysis to the extent that the psychologic method inaugurated by Freud differs from that of other medical disciplines. Patients who are entering analytic treatment and their families have always to be informed of the regulations which the psychoanalytic technic impose and of the reasons for their existence. This is tedious work—an expenditure of time and energy on the part both of the patient and of the analyst. Dr. Kubie's book is written to curtail this essential introduction and to make it possible for the actual analysis to begin the sooner. It is a book which might well be given every prospective patient for psychoanalytic treatment, as well as the responsible members of the patient's family. In addition, the book will prove valuable to physicians who are familiar with psychoanalytic theory but who may fail to see why analysis should be carried on under conditions so foreign to ordinary medical practice. In the reviewer's opinion, this is one of the few books written for laymen which will also be of profit to many physicians.

Dr. Kubie's book is written to explain such matters as the usual requirement of daily sessions, procedures adopted to secure free association, matters of finance, importance of the rôle of family and friends, etc. The author also gives information concerning the training of analysts and the possible methods of selecting one. Relatively few persons realize that the training for the practice of analysis has now become as systematized as the preparation for any other medical specialty. It is a costly, time-consuming, elaborate process, which distinguishes the freudian analyst from the pupils of other schools of psychotherapy, which as yet have little, if any, formalized training.

In presenting the various practical aspects of psychoanalysis, the author has found it expedient to introduce some remarks on theory, although this is not his chief concern. His summary is noteworthy and is therefore quoted: "(1) There are unconscious mental forces. (2) These always play an important rôle in determining human behavior. (3) Where they play a *dominant* rôle, such behavior cannot be influenced materially without altering these underlying factors. (4) In order to do this, it is first necessary to find out what the unconscious mental forces are. (5) This makes necessary the use of a highly specialized technique, which is designed to overcome certain obstacles to the exposure of unconscious material. (6) This technique is at the same time effective in modifying the influence of the unconscious forces which are brought to light."

Dr. Kubie's statements are clarified and enlivened by a happy choice of illustrative material from his own practice and that of his colleagues. He is careful to use this method also in his endeavor to present the causes of failure in analytic therapy.

It is important to note that this book was originally conceived as an aid to Dr. Kubie himself in dealing with the numerous inquiries made by prospective patients, their families and physicians. His colleagues, feeling a similar need, have welcomed the book. There is no intention to propagandize or to sift the philosophic bases of the theory. However, Dr. Kubie has stated some of the accepted theories by way of explanation of practical matters in such an illuminating manner that his book may have an even larger application than the one for which it was intended.

Elements of Psychology. By Knight Dunlap. Price, $3. Pp. 499. St. Louis: C. V. Mosby Company, 1936.

This book is the result of Dunlap's revision of his earlier work "Elements of Scientific Psychology," published in 1922, but, as he indicates in the preface, it is essentially a new book. It is intended primarily as an introductory textbook for college students and should serve this purpose admirably. The biologic point

of view of the author is well set forth in his definition of psychology, as "the study of life as the total activity of the individual animal." Although the subject matter discussed includes the usual topics of systematic psychology, such as the senses, perception, learning, feeling and affect and so on, the concept of the total personality is stressed throughout. These phenomena are presented not as isolated activities but as synonymous with the experiencing of the living organism. This biologic approach is the outstanding feature of the book which distinguishes it from other texts. What can be observed, the author maintains, are human bodies and their actions. Sensing, perceiving, feeling and so on are acts of being conscious and as such cannot be observed directly. Nevertheless, the author differs from the radical behaviorists who would eliminate consciousness from their subject matter. Although psychologists study "a body which acts," the body in action is synonymous with the cooperative activity of the sense organs, nervous system and muscles. Consciousness depends for its organic basis on these activities and cannot be separated from them. The inadequacy of the stimulus-response type of psychology is further emphasized in that a response is considered not merely as a contraction of a group of muscles or the secreting of a gland but as a total integrated response which includes all the cellular activities that result from a stimulus. The final chapter, on maladjustment and readjustment, offers a simplified classification of mental disorders into psychoses (organically determined) and neuroses ("bad habits of response"). The psychoses are further divided into definite psychoses (those of known cause) and probable psychoses (those of unknown cause). Throughout the book extreme care is exercised in defining terms precisely. A glossary serves to bring these definitions together in a convenient form.

Saggio di fisiologia del liquido cerebro-spinale (Critical Essay on the Physiology of the Cerebrospinal Fluid). By Beppino Disertori, University of Milan. Price, 24 lire. Pp. 178, with 22 illustrations and 5 tables. Rome: Luigi Pozzi, 1935.

In this monograph Disertori attempts to analyze critically the concepts regarding the method of formation of the cerebrospinal fluid. It is divided into ten chapters of one hundred and sixty-one pages, with a bibliography of fifteen pages. The site of formation, the circulation and the function of the fluid are dealt with in the last three chapters, and the usual ideas in regard to these subjects are expressed.

The first chapter discusses the two most commonly considered theories of the mode of formation of the fluid, namely, dialysis and secretion. Disertori states that the cerebrospinal fluid cannot be considered to be a dialysate of the serum unless the concentration of the various anions and cations in the two fluids satisfies the Donnan theory of membrane equilibrium. In the five succeeding chapters he discusses the distribution between the serum and the cerebrospinal fluid of chlorides, bromides, dextrose, other substances normally present in the blood and foreign substances introduced into the blood.

Disertori concludes that the Donnan theory of membrane equilibrium is not satisfied by the distribution of any of these substances between the serum and the cerebrospinal fluid and that, therefore, the cerebrospinal fluid must be considered as a secretion. The arguments are based on an incomplete review of the literature and an analysis of the serum and cerebrospinal fluid of sixty-eight patients personally examined, with the examination of the first and last specimens of fluid removed at encephalography in forty cases. This number of cases is obviously too small to form a basis for the author's conclusions; also, the arguments advanced to support his conclusions are not presented in a new or different fashion. Therefore, unfortunately, the monograph does not lead any nearer to a settlement of the controversy on secretion versus dialysis.

Syphilis and Its Treatment. Macmillan Medical Monographs. By William A. Hinton, M.D. Price, $3.50. Pp. 321. New York: The Macmillan Company, 1936.

The author's purpose in writing this book was to provide a clear, simple and relatively complete account of syphilis and its treatment for students, general practitioners and public health workers. The book is divided into three parts: (1) manifestations of syphilis, (2) treatment and (3) laboratory technic.

The book is written in an interesting style, and most of the facts in relation to syphilis are covered, although many rather inadequately. For the neurologist, who deals with involvement of the nervous system by syphilis, the book will have little value. Only seventeen pages are given to the symptomatology of neurosyphilis, five to treatment of neurosyphilis and seventeen to examination of the spinal fluid. The author's lack of clinical experience, especially with neurosyphilis and other diseases of the nervous system, is shown by such dogmatic statements in regard to diagnosis as that paralysis of the third nerve "as an acquired sign is almost pathognomonic of neurosyphilis" (page 132) or that "a negative colloidal gold test is the best single evidence that the disturbance of the central nervous system is functional and not organic" (page 140). Unfortunately, also, the author has given too prominent a place to his prejudices against the advisability and diagnostic value of lumbar puncture and special methods of treatment. In discussing the treatment for neurosyphilis, he recommends administration of mercury by mouth or by inunction and the use of arsphenamine. He mentions that tryparsamide and fever therapy are used by many neurologists in the treatment for dementia paralytica but he is skeptical that the results obtained are due to these methods of therapy, since, according to him, it is not known in what measure the good results are due to previous or subsequent injections of bismuth or arsphenamine.

The position taken by Hinton in regard to spinal puncture and the treatment for neurosyphilis, which is in discord with generally accepted views, seriously lessens the value of the book. In addition to these defects, the author recommends in the therapy for early syphilis the use of mercury by mouth or inunctions as treatment between the courses of arsphenamine, although it is recognized that mercury has a low index of therapeutic efficiency. It is unfortunate that the author has allowed his prejudices to detract from the value of an otherwise well presented work.

Handbuch der Neurologie. Edited by O. Bumke and O. Foerster. **Volume IV. Allgemeine Neurologie. Teil IV: Hirnnerven. Pupille.** Price, 135 marks. Pp. 701, with 173 illustrations. Berlin: Julius Springer, 1936.

This new volume of the *Handbuch* by Bumke and Foerster deals entirely with the cranial nerves. It is an exhaustive consideration of the subject. Riese contributes a brief consideration of diseases of the olfactory nerves and cortex, about which not much is known. There is a lengthy and good discussion of the symptomatology of diseases of the optic nerve by Marchesani, including choked disk, optic neuritis and atrophy of the optic nerve. There is a useful article by Jaensch on the methods of investigation of the optic nerve. One of the best articles is that by Bielschowsky on the symptomatology of the oculomotor apparatus. This is a clear, concise and yet detailed description of the various disorders arising from oculomotor disability. Jaensch has a lengthy contribution on the pupils, covering fully the various pupillary disorders. There is a short chapter by Kramer on diseases of the fifth, seventh, ninth, eleventh and twelfth cranial nerves. Klestadt contributes a long chapter of almost two hundred pages on the auditory nerve and its diseases, which is a splendid review of the subject.

The volume maintains the high standard of excellence created by the earlier volumes of this *Handbuch*. It is carefully done, well illustrated and exhaustive in its consideration of the subject-matter. It is recommended without hesitation to all neurologists.